*UNIVERSITE MONTPELLIER II*
*SCIENCES ET TECHNIQUES DU LANGUEDOC*

***T H E S E***

pour obtenir le grade de

**DOCTEUR DE L'UNIVERSITE MONTPELLIER II**

***Discipline : Microbiologie, Parasitologie***

***Ecole Doctorale : SIBAGHE***

Présentée et soutenue publiquement

par

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Le 19 juillet 2011

***Titre :***

# Surveillance et contrôle de la Peste des Petits Ruminants : apports de la modélisation

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**TITRE** : Surveillance et contrôle de la Peste des Petits Ruminants : apports de la modélisation

**RESUME** : La Peste des Petits Ruminants est une maladie contagieuse virale négligée affectant principalement les caprins et les ovins et qui continue son expansion malgré l'existence de moyens de diagnostic et de vaccins efficaces. Le développement d'approches épidémiologiques et économiques avec des outils d'aide à la décision tels que les modèles semble indispensable dans ce contexte si on vise à l'amélioration de sa surveillance et de son contrôle, ainsi qu' à la mobilisation des bailleurs. L'objectif de ce travail est d'y apporter une contribution originale dans un contexte africain où les données ne sont pas toujours disponibles ou faciles à collecter. Plusieurs approches de modélisation complémentaires sont abordées dont un modèle déterministe à compartiments (SEIR), un modèle de régression logistique et un modèle basé sur la théorie des réseaux sociaux. La pertinence de très hauts niveaux de vaccination et d'une surveillance active par sérologie telle qu'habituellement préconisée dans les pays du 'Sud', dont les systèmes de production sont les seuls véritablement menacés, est discutée. Dans le cas de l'Ethiopie, un système de surveillance passif syndromique est envisagé avec une concentration possible des efforts de sensibilisation à la maladie au niveau des points de pâturages. Concernant la répartition temporelle et spatiale du niveau vaccinal à appliquer, la mise en place de 'barrières' vaccinales en lien avec la géographie du pays est suggérée comme pouvant optimiser la pratique actuelle de vaccination en urgence autour des foyers déclarés lorsque les ressources sont disponibles. L'intégration de l'écologie de la maladie et l'utilisation complémentaire aux modèles mathématiques de l'analyse phylogéographique offrent des perspectives intéressantes mais restent encore un défi ; la prise en compte de critères socio-économiques est par contre une priorité pour parfaire notre approche.

**MOTS-CLES** : PPR, Modélisation, Surveillance, Contrôle, Epidémiologie, SNA, Ethiopie

**TITLE** : Peste des Petits Ruminants surveillance and control : Use of modeling

**SUMMARY**: Peste des Petits Ruminants (PPR) is a neglected viral contagious disease of sheep and goats. It has a widespread distribution that continues to expand despite good diagnostic tests and vaccines. Considering this and with the aim to improve surveillance and control of the disease and to attract funding for this, it would be necessary to develop epidemiological and economic approaches including decision tools such as models. The objective of this work is to contribute to such improvements in an African context where data are hardly available or collecting them is a challenge. Various complementary modeling approaches are reported among which a compartmental model (SEIR), a logistic model and a model based on social network theory. The relevance of very high vaccination levels and of active surveillance based on serology as usually recommended worldwide is discussed for developing countries which are the only ones truly threatened by PPR. In the case of Ethiopia, a passive syndromic surveillance system is being considered, enhancing disease awareness at grazing points. Regarding the spatial and temporal distribution of the vaccination level to be administered, ring vaccination making the best use of the country's topography is suggested to enhance effectiveness of the actual practice that consists of outbreak emergency vaccination when resources are available. Including the ecology of the disease and linking phylogeographical analysis to the existing mathematical models offers interesting perspectives but remains a challenge. However, taking into account socio-economic criteria should be a priority to fine-tune our approach.

**KEY-WORDS**: PPR, Modeling, Surveillance, Control, Epidemiology, SNA, Ethiopia

# Remerciements

A Serge Morand, directeur de cette thèse pour son soutien infaillible quelque soit le projet, ses précieux conseils scientifiques et personnels.

A Véronique Chevalier pour avoir accepté d'être co-directeur de thèse.

A Alain Milon d'avoir accepté de présider ce jury.

A Dirk Berkvens et Christian Ducrot de me faire l'honneur d'être rapporteurs pour cette thèse et Adama Diallo de faire partie du jury.

A François Roger sans qui ce manuscrit ne serait pas ce qu'il est. Merci de m'apporter ton excellence scientifique. Merci aussi de ta clairvoyance, de la confiance et de la liberté que tu as su m'accorder.

A Geneviève Libeau pour ses remarques pertinentes au cours de ces travaux.

A Guy-Pierre Martineau, Jean-Luc Guérin, Stéphane Bertagnoli, mes supers 'coachs' de l'ENVT.

A Flavie, Aurélie, Mathilde pour nos discussions constructives et nos franches rigolades.

A l'équipe AGIRs mais plus particulièrement à Marie, magicienne de la cartographie.

# TABLE DES MATIERES

# INTRODUCTION

La Peste des Petits Ruminants (PPR) est une maladie contagieuse affectant principalement les caprins et les ovins. Elle est due à un virus de la famille des *Paramyxoviridae*. Malgré une première description en 1942 en Côté d'Ivoire, il est probable que la maladie ait été présente bien avant, étant difficile à distinguer de la Peste Bovine ou confondue avec des pathologies pulmonaires de surinfections bactériennes (Taylor et al., 2002). Maladie sévère à dissémination rapide, elle est aujourd'hui reconnue comme responsable de pertes à travers la plupart des pays de l'Afrique sub-saharienne au nord de l'équateur, dans la péninsule arabique, en Inde et dans beaucoup d'autres pays en Asie (Lefèvre and Diallo, 1990 ; Shaila et al., 1996 ; Diallo, 2003 ; Gopilo, 2005 ; Kwiatek et al., 2007). Malgré l'existence de moyens de diagnostic et de moyens de contrôle efficaces (vaccin avec protection pendant toute la vie économique de l'animal), la maladie continue son expansion et menace les systèmes de production de petits ruminants particulièrement importants dans les pays en voie de développement dont une partie de l'Afrique, constituant le danger le plus important pour la survie des petits éleveurs (Empres, 2009).

Le développement d'approches épidémiologiques et économiques quantitatives avec notamment des outils d'aide à la décision tels que les modèles semble indispensable dans ce contexte pour l'amélioration du contrôle de la PPR et la mobilisation des bailleurs. Il en est de même pour l'amélioration de la surveillance étroitement liée à celle du contrôle.

La surveillance sera définie ici comme la collection continue et systématique d'informations utiles concernant une maladie, une infection ou le bien-être dans une population animale déterminée, intégrée avec la communication à temps de résultats intéressants aux personnes concernées y compris les responsables des mesures de contrôle et de prévention (Stärk et al., 2002 ; Thurmond, 2003). La rationalité de la surveillance de la PPR est de rapidement détecter de nouveaux foyers - une détection précoce permettant une prise de décision plus efficace (réaction rapide) - de révéler des changements d'incidence ou de prévalence de la maladie dans les zones infectées et vaccinées et de certifier l'absence de maladie. Il apparait essentiel de renforcer et de continuer de développer les systèmes de surveillance dans les pays et régions affectés par la PPR ou à risque accru de la maladie et de

déterminer des stratégies de contrôle optimum pour développer une meilleure gestion du risque.

L'objectif de ce travail est d'apporter une contribution originale à ces problématiques en intégrant des études épidémiologiques et des approches de modélisation dans un contexte africain où les données ne sont pas toujours disponibles ou faciles à collecter. La valorisation des informations disponibles permet de simuler la transmission de la maladie entre espèces réceptives et l'effet de différents schémas de vaccination, d'évaluer la probabilité d'occurrence spatiale de la maladie dans un grand pays d'élevage, l'Ethiopie, avec les facteurs de risque associés, et enfin de déterminer des points de contact entre animaux et troupeaux à considérer pour mieux cibler la surveillance et la lutte.

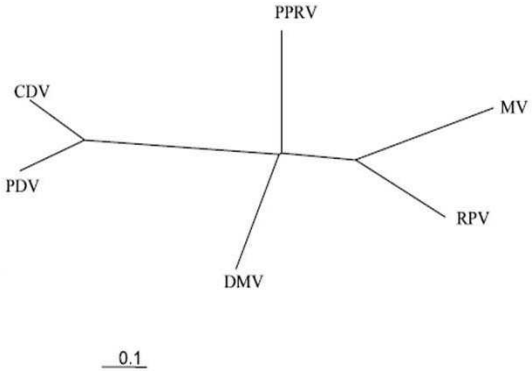


**Figure 1 : Arbre phylogénétique des morbillivirus (Source : Mahapatra et al., 2003).** La taille des branches est proportionnelle aux distances génétiques selon l'échelle indiquée – l'échelle de 0,1 indique 0,1 substitution nucléotidique par site. Légende : MV : measles virus (rougeole), RPV : rinderpest virus (peste bovine), DMV : dolphin morbillivirus (morbillivirus du dauphin), PDV : phocin distemper virus (morbillivirus du phoque), CDV : canine distemper virus (maladie de Carré), PPRV : virus de la peste des petits ruminants.

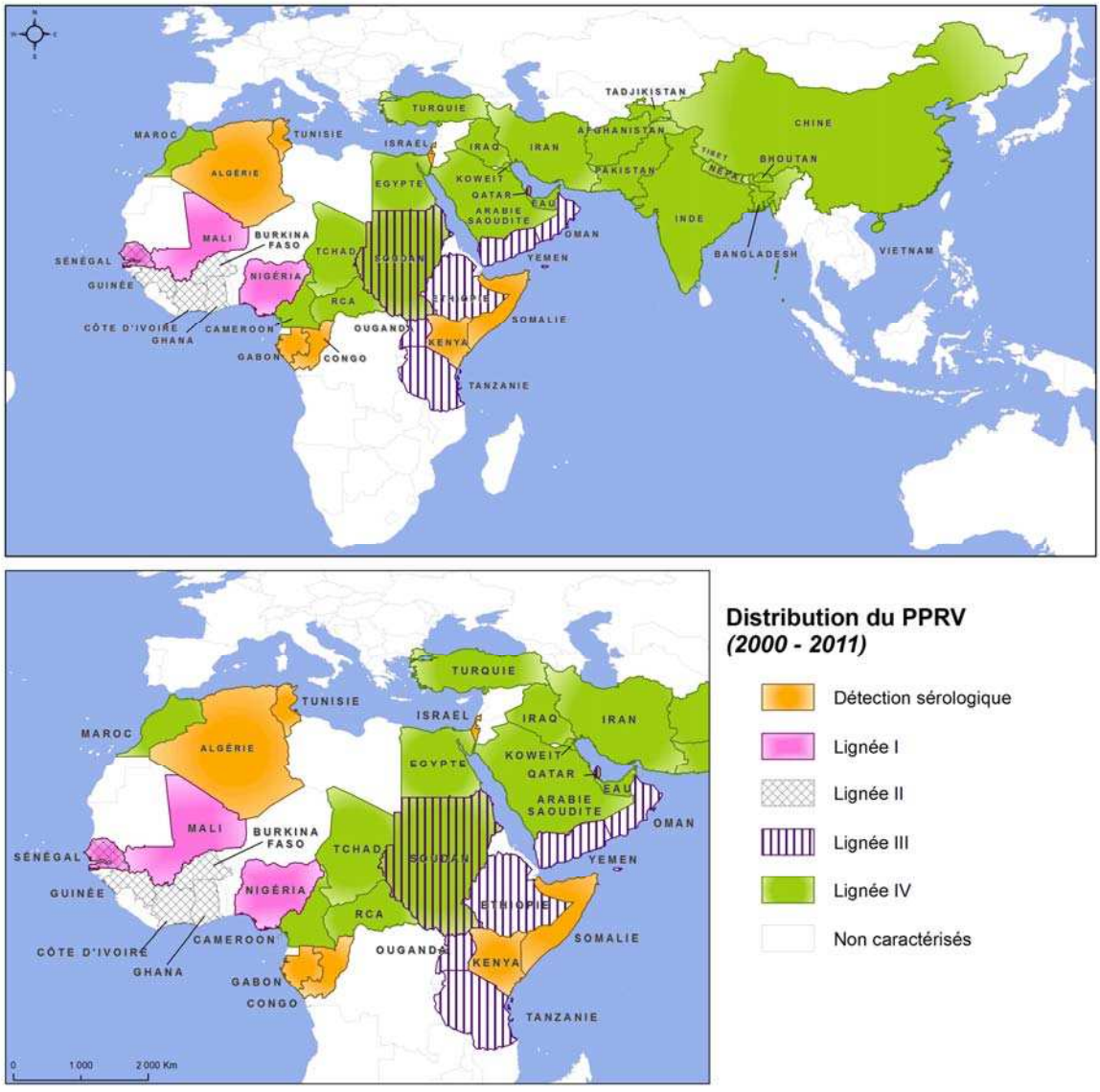


**Figure 2 : Carte de la distribution des différentes lignées de PPRV (Source : adaptée de Banyard et al., 2010)**

# I. CONTEXTE THEORIQUE ET OBJECTIFS DE LA THESE

## I.1 LA PESTE DES PETITS RUMINANTS : UNE MALADIE A FORT IMPACT EN EXPANSION

### I.1.1. Définition, étiologie, clinique

La PPR est causée par le virus PPRV. Ce virus appartient au groupe des *Morbillivirus* de la famille des *Paramyxoviridae* (Gibbs et al., 1979 ; Saliki, 1998). Il est étroitement associé au virus de la peste bovine des buffles et du bétail (RPV), le virus de la rougeole humaine (MV), le virus de la maladie de Carré des chiens et de certains carnivores sauvages (CDV) et de *Morbillivirus* de mammifères marins (PDV, DMV) (Barrett et al., 1993 ; Jones et al., 1993 ; Scott, 1981 ; Yayehrad, 1997) (Figure 1). Une des principales caractéristiques de ces virus est la sévère immunosuppression transitoire qu'ils induisent chez leurs hôtes respectifs, favorisant ainsi des infections parasitaires et bactériennes secondaires comme dans le cas du PPRV par exemple (Adombi et al., 2011 ; Abubakar et al., 2011). Contrairement au virus de la rougeole on ne dispose pas d'information sur la période d'origine du PPRV et sur son éventuel dichotomie du virus de la peste bovine qui pourrait s'être adapté aux petits ruminants (Furuse et al., 2010 ; Libeau, communication personnelle).

Il n'y a pas de variation notable dans la pathogénicité du virus (Taylor, 1984). Certaines souches montrent de légères variations dans le schéma de migration par électrophorèse de leurs protéines mais sans relation avec la pathogénicité qui leur est associée (Diallo A. *et al.*, 1987). Des éléments récents suggèrent néanmoins une évolution de pathogénicité qui pourrait peut-être être associée à l'apparition de nouvelles souches (Khalafalla et al., 2010). On distingue actuellement quatre groupes (Figure 2). La lignée I en Afrique de l'Ouest qui regroupe les isolats des années 70 et d'autres plus récents d'Afrique Centrale; la lignée II avec les isolats de Côte d'Ivoire, de Guinée et du Burkina, la lignée III en Afrique de l'Est, au Soudan, Yémen et en Oman et enfin la lignée IV qui inclut les virus isolés des foyers récents dans la péninsule arabique, le moyen orient, l'Asie du sud et récemment de certains

territoires africains (Banyard et al., 2010 ; Kwiatek et al., 2011). Il n'y a pas d'élément aujourd'hui de filiation des lignées les unes par rapport aux autres, les voies de migration des animaux d'Est en Ouest pouvant cependant laisser suggérer que les lignées africaines dériveraient de la lignée asiatique (Libeau, communication personnelle).

La PPR est une maladie sévère à dissémination rapide sur les petits ruminants. Elle est caractérisée, pour les formes aigües, par l'apparition soudaine de dépression, de fièvre, d'écoulement nasal et oculaire, d'une diarrhée nauséabonde et de la mort. C'est une maladie de l'ex liste A de l'Organisation Mondiale de la Santé Animale (OIE) (Lefevre and Diallo, 1990 ; Agriculture and Resource Management Council of Australia and New Zealand, 1996), désormais incluse dans le groupe des maladies animales importantes du point de vue économique et devant être notifiée. La durée de la maladie est de 5-10 jours avec une période d'incubation de 3 à 5 jours (Lefevre and Diallo, 1990 ; Braide, 1981 ; Taylor, 1984 ; Diallo, 2004) allant même jusqu'à 10 jours (OIE, 2005) pendant laquelle les animaux peuvent transmettre la maladie (Saliki, 1998). Les taux de morbidité peuvent varier de 10 à 80% et les taux de mortalité de 0 à 90% (Akakpo et al., 1996 ; Nanda et al., 1996 ; Rossiter and Taylor, 1994 ; Wakwaya, 1997 ; Diallo, 2003 ; OIE, 2005 ; Tesfaye, 2005).

Le diagnostic de laboratoire est indispensable pour établir un diagnostic de certitude. De nombreuses techniques sont décrites pour la détection de l'antigène, l'isolement et l'identification du virus, la détection de l'acide ribonucléique (ARN) viral ou enfin celle des anticorps (Banyard et al., 2010).

### I.1.2. Histoire naturelle de la PPR

*Espèces réceptives et sensibles*

Si les moutons et les chèvres sont réceptifs, les chèvres sont considérées comme plus sensibles, d'une part parce que dans de nombreux rapports la maladie n'est mentionnée que sur les chèvres, et d'autre part parce que les chèvres sont souvent atteintes sans que les moutons vivant à proximité le soient (Lefèvre et Diallo, 1990 ; Diallo, 2003 ; Chauhan et al., 2009 ; Roeder et al., 1994 ; Singh et al., 2004 ; Taylor and Abegunde, 1979 ; Wang et al., 2009 ; Abubakar et al., 2011). Cependant, il a été signalé des cas de PPR où les moutons ont payé de lourds tributs par rapport aux chèvres notamment en Asie, en Ethiopie mais aussi

plus récemment au Maroc (Abraham et al., 2005 ; Direction de l'élevage au Maroc, 2008). Des foyers où seuls les moutons étaient atteints ont aussi été rapportés (Yesilbag et al., 2005 ; Roger, communication personnelle). La race et l'âge jouent aussi un rôle prépondérant dans la sensibilité au virus, les jeunes de 4-12 mois étant plus sensibles (Diallo, 2003 ; OIE, 2005 ; Diop et al., 2005 ; Gopilo, 2005).

Le rôle de la faune sauvage dans l'épidémiologie de la PPR n'est pas établi (Banyard et al., 2010). Ce rôle semble pourtant important même si aucun cas n'a encore été observé sur le terrain. Un foyer de PPR a été décrit sur des gazelles et des daims d'un parc zoologique (Furley et al., 1987), ainsi que sur d'autres petits ruminants sauvages comme des moutons de Laristan, des gazelles Dorcas, des gazelles gemboks, des gazelles de Thomson, des bouquetins de Nubie (Diallo, 2003 ; Abu Elzein, 2004), des céphalophes de Grimm (Ogunsanmi et al., 2003) et des impalas (Kinne et al., 2010).

Les grands ruminants, les vaches, les buffles (*Bubalus bubalis*, *Syncerus caffer*) et les porcs peuvent être infectés (production d'anticorps) mais il y a peu voire aucune évidence de la maladie associée à cette infection (Govindarajan et al., 1997 ; Couacy-Hymann et al., 2005). Le rôle joué par les bovins dans la circulation du virus reste encore imprécis (Saliki, 1998). Quand on inocule le virus au bétail, il développe une hyperthermie transitoire qui passe souvent inaperçue, suivie d'une séroconversion qui lui procure une solide protection contre une exposition au virus de la peste bovine (Hamdy et al., 1976). Considérant l'effet immunosuppressif du PPRV comme d'autres *Morbillivirus*, il serait possible qu'en fonction de l'âge ou du statut physique de l'animal hôte, le virus surpasse occasionnellement la résistance innée des grands ruminants et induise des signes cliniques similaires à la peste bovine. Si cette hypothèse est vérifiée cela constitue un risque sérieux pour le bétail localisé dans les zones endémiques de PPR qui n'est plus vacciné par le vaccin peste bovine et donc indirectement protégé, compte tenu des contraintes imposées par l'avancée mondiale vers l'éradication imminente de cette maladie (Diallo et al., 2007).

Les dromadaires apparaissent réceptifs (Abraham et al., 2005 ; Abubakar et al., 2008 ; Albayrak and Gür, 2010, El Amin and Hassan, 1998 ; Haroun et al., 2002) et sensibles à la maladie (Roger et al., 2000, 2001). Le rôle possible des dromadaires dans la transmission du PPRV aux petits ruminants a été suggéré en Ethiopie (Roger et al., 2001) puis en Arabie

Saoudite (El-Hakim, 2006). De récentes enquêtes au Soudan n'ont pas permis de confirmer ou infirmer cette possibilité de transmission (Khalafalla et al., 2010).

La classification des hôtes en catégories telles que définies en écologie de réservoir, de 'spillover' ou d'aberrant peut aider à une meilleure description des dynamiques de l'infection au sein d'un patho-système (Morris and Jackson, 2005). La séparation entre les différents types d'hôtes n'est pas fixe et une espèce hôte peut changer de catégorie suite à un changement de structure génétique du virus ou un changement d'écologie de l'hôte. Un hôte réservoir est un hôte qui maintient l'infection et ne contracte généralement pas la maladie ou bien dont les signes cliniques sont peu sévères ou enfin dont seul les jeunes animaux sont atteints cliniquement alors que les adultes sont immuns ou infectés de manière sub-clinique. C'est le cas des moutons et des chèvres pour la PPR, peut-être celui des dromadaires. Les connaissances actuelles appellent cependant à la prudence pour beaucoup d'espèces dans le cas de la PPR et notamment pour les dromadaires. En effet, les dromadaires pourraient tout aussi bien être des hôtes 'spillover'. Un hôte 'spillover' est sensible à l'infection et excrète l'agent pathogène pouvant ainsi transmettre l'infection à d'autres hôtes. Cependant, il ne peut maintenir l'infection au sein de son espèce sur le long terme sauf s'il y a un apport constant ou intermittent d'infection depuis un hôte réservoir (Morris and Pfeiffer, 1995 ; Corner, 2006). Ainsi, si les échanges infectieux avec les hôtes réservoirs sont éliminés, l'infection s'éteindra à plus ou moins long terme au sein des hôtes 'spillover'. Certaines espèces de la faune sauvage ou les bovins pourraient aussi faire partie de cette catégorie bien que ces derniers semblent plutôt appartenir aux hôtes aberrants. Les hôtes aberrants ne sont infectés que rarement, expriment généralement des signes cliniques sévères et n'excrètent généralement pas assez de virus pour le transmettre à d'autres hôtes. Ils n'ont donc pas une grande importance dans le cycle épidémiologique de la maladie mais peuvent être sévèrement atteints.

*Voies de transmission : directes vs. indirectes*

La PPR se transmet principalement par contact direct étroit (Braide, 1981 ; OIE, 2005 ; Saliki, 1998), le virus est excrété dans les sécrétions oculaire et nasale ainsi que dans les fécès des

animaux malades (Bundza et al., 1988, Ezeibe et al., 2008). Le virus fut aussi retrouvé dans la salive et l'urine.

La transmission indirecte semble être difficile compte tenu de la faible résistance du virus dans l'environnement et sa sensibilité aux solvants lipidiques (Lefèvre and Diallo, 1990 ; Diallo, 2003). Il n'existe aucun stade de porteur chronique (Saliki, 1998 ; Diallo, 2004 ; OIE, 2005 ; Gopilo, 2005).

*Patterns spatio-temporels*

La PPR fut décrite au départ en Afrique de l'Ouest (Gargadennec and Lalanne, 1942), mais la maladie est aujourd'hui reconnue comme responsable de mortalité et de morbidité à travers la plupart des pays de l'Afrique sub-saharienne au nord de l'équateur, dans la péninsule arabique, en Inde et dans beaucoup d'autres pays en Asie (Lefèvre and Diallo, 1990 ; Shaila et al., 1996 ; Diallo, 2003 ; Gopilo, 2005 ; Kwiatek et al., 2007) (Figure 2). Le virus fut isolé au Sénégal, en Côte d'Ivoire, en Guinée (Shaila et al., 1996), au Nigéria (Taylor and Abugunde, 1979 ; El-Yuguda et al. 2010 ; Ibu et al., 2008), au Burkina (Sow et al., 2008) mais aussi au Ghana (Banyard et al., 2010). En Afrique de l'Est, la PPR est considérée comme endémique, confirmé par la détection d'anticorps contre le PPRV au Kenya (1999 et 2009) et en Ouganda (2005 et 2007) (Banyard et al., 2010). Le virus fut isolé au Soudan (Elhag Ali and Taylor, 1984 ; Saeed et al., 2010) et récemment en Tanzanie (2010) après que sa transmission sur le terrain ait été confirmée (Swai et al., 2009). Des sérologies positives ont été rapportées en Afrique Centrale : République Centrafricaine (1999, 2005 et 2006), Congo (2006), Tchad (1999 et 2006), Cameroun (2009) et Gabon (2007) (Banyard et al., 2010). En Afrique du Nord le PPRV avait été détecté historiquement en Egypte suivi d'une résurgence du virus en 2006 jusqu'à ce que d'importants foyers se déclarent au Maroc en 2008 et que des évidences sérologiques d'infection soient rapportées en Tunisie (Ayari-Fakhfakh et al., 2010) suggérant que le virus puisse être présent dans d'autres régions nord africaines. En Asie, des foyers furent signalés en Turquie (Ozkul et al., 2002 ; Yesilbag et al., 2005 ; Tufan, 2006 ; Kul et al., 2007 ; Albayrak and Alkan, 2009) et en Arabie Saoudite (Abu Elzein et al., 1990 ; Housawi et al., 2004 ; Al Dubaib 2008, 2009 ; Abu Elzein et al., 2004 ; El Rahim et al., 2005). Des séroprévalences furent rapportées en Jordanie (Al-Majali et al. 2008), au Liban (Attieh,

2007) ; le virus isolé aux Emirats Arabes Unis (Kinne et al., 2010) et au Qatar (Banyard et al., 2010). Le virus circule aussi au Yémen, au Pakistan (Mahmood et al., 2009 ; Durrani et al., 2010 ; Zahur et al, 2009) et est endémique en Inde (Shaila et al., 1989 ; Dhar et al., 2002 ; Kataria et al., 2007 ; Saha et al., 2005 ; Chavran et al., 2009 ; Santhosh et al., 2009 ; Raghavendra et al., 2008). Au Moyen Orient des foyers sont régulièrement identifiés en Iraq (Barhoom et al., 2010) et en Iran (Abdollapour et al., 2006). La récente détection du PPRV au Proche Orient a surligné son potentiel de diffusion dans des régions qui n'avaient pas été touchées jusque là, avec une présence au Tadjiskistan (Kwiatek et al., 2007) et au Kazakhstan (Lundervold et al., 2004). En Chine de récents foyers ont été déclarés au Tibet en juillet 2007 (Wang et al., 2009) suggérant que le virus puisse être présent de manière beaucoup plus étendue que l'on ne croit. Ainsi il est possible que le PPRV ait diffusé dans beaucoup d'autres pays voisins mais qu'il passe inaperçu par méconnaissance des populations locales ou soit confondu avec d'autres maladies aux manifestations cliniques similaires (Banyard et al., 2010). Le faible effectif de petits ruminants ainsi que leur faible importance économique et culturelle dans la plupart des pays d'Asie du sud-est pourraient aussi expliquer que la maladie n'y ait été rapportée qu'une fois.

En général la PPR sévit sous forme de foyers épizootiques cycliques et saisonniers (Figure 3). En Afrique on a une augmentation du nombre de foyers durant la saison froide, au début de la saison des pluies. Différentes hypothèses sont avancées pour expliquer ce caractère saisonnier :

- Pendant la saison froide le temps de survie du virus est probablement plus long (surtout pendant la nuit) et les possibilités de contamination d'un grand nombre d'animaux sont plus importantes. Par ailleurs le froid constitue un stress pour les animaux et une diminution de leur résistance.

- Les précipitations du début de saison des pluies peuvent aussi constituer un stress pour les animaux qui, déjà affaiblis par une longue période de sécheresse (peu de nourriture), voient leur résistance diminuée.

- A l'approche de certaines fêtes religieuses le commerce de ces animaux s'intensifie et leurs attroupements extrêmement importants sur les marchés constituent des

conditions idéales de transmission du virus. Les sujets contaminés à ce moment vont propager l'infection aux troupeaux dans lesquels ils seront nouvellement introduits en attendant le sacrifice.

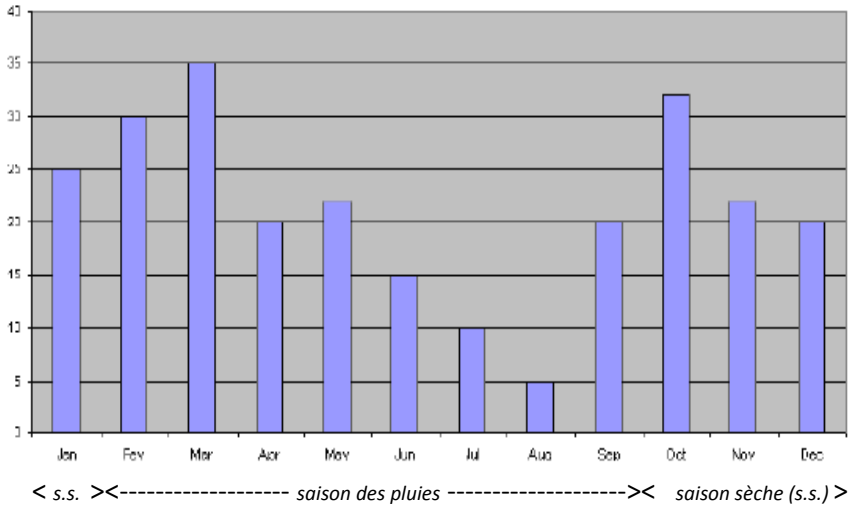


**Figure 3: Allure saisonnière de la maladie dans les endroits où elle est endémique (Source : Gopilo , 2005) : Nombre de foyers de maladie en fonction des mois de l'année.**

Outre son caractère saisonnier on constate qu'elle évolue sous un mode cyclique (3 ans en moyenne). Ceci s'explique par le fait que les animaux ayant survécu à la PPR sont protégés à vie. Cette immunité acquise est solide par rapport à une réinfection éventuelle. La durée de cette protection n'est pas établie mais il s'agit sans doute de la vie économique entière de l'animal. De ce fait, le troupeau ne peut connaître une nouvelle épizootie qu'après le renouvellement des individus qui le composent. Or 90 à 100% des petits ruminants sont remplacés en 3 ans, ce qui entraîne la constitution de troupeaux d'animaux sensibles, situation à nouveau favorable à l'apparition de la maladie (Diallo, 2003 ; Saliki, 1998).

### I.1.3. Déterminants

*Facteurs de risque*

En l'absence d'étude épidémiologique analytique de la PPR, un ensemble de facteurs de risque potentiels, d'après la littérature, est présenté dans le Tableau 1.

*Facteurs protecteurs et contrôle*

Il n'y a pas de traitement spécifique pour les animaux atteints. Certains auteurs ont préconisé l'administration de sérum anti-PPR ou d'antibiotiques ou encore de traitements anti-diarrhéiques. Mais de tels traitements n'ont sans doute pas beaucoup d'intérêt en pratique dans les conditions de terrain compte tenu des prix d'un individu mouton ou chèvre (Diallo, 2004).

Tous les moutons et les chèvres d'un troupeau atteint devraient être placés en quarantaine jusqu'à au moins un mois après le dernier cas clinique (Diallo, 2004). Les mouvements des animaux doivent être strictement contrôlés dans la zone de l'infection (Agriculture and Resource Management Council of Australia and New Zealand, 1996).

Malheureusement toutes ces mesures sanitaires sont difficiles à maintenir dans tous les pays où la PPR est endémique (Tesfaye, 2005 ; Singh et al., 2009).

Pendant longtemps la vaccination fut pratiquée à l'aide d'un vaccin hétérologue préparé à l'aide du virus atténué contre la peste bovine profitant de la protection croisée entre les deux morbillivirus due à une communauté antigénique étroite (Couacy-Hymann et al., 1995). Il a été prouvé qu'il apportait une protection pour au moins 1 an et probablement pour la vie économique des animaux vaccinés (Patout, 1995). Dans le cadre des programmes d'éradication de la peste bovine, et pour ne pas gêner les dépistages sérologiques de cette maladie chez tous les animaux sensibles au virus bovipestique, l'utilisation de ce vaccin hétérologue est interdite. En Inde trois vaccins vivants atténués sont autorisés: Sungri 96, Arasur 87 et Coimbatore 97 (Saravanan et al., 2010). En Afrique, c'est le vaccin homologue développé en 1989 (FAO, 1997), atténué par passage en série sur les cellules VERO qui est utilisé à la dose de $10^{2,5}$ $DICT_{50}$ de virus par animal en injection sous-cutanée (Diallo et al., 1989 ; Martrenchar, 1999 ; Diallo, 2003). Il procure une immunité à vie contre la PPR chez les animaux inoculés et peut protéger les chèvres contre une contamination par un virus virulent de peste bovine (Couacy-Hymann et al., 1995). Son innocuité sur les chèvres gestantes quel que soit le stade de la gestation a aussi été prouvée. En outre des anticorps anti-PPR colostraux ont été retrouvés chez des chevreaux jusqu'à l'âge de 3 mois (FAO, 1997).

Sa faible thermostabilité peut cependant en limiter l'efficacité. D'autre part, les animaux ayant reçu ce vaccin ne peuvent pas être différenciés sérologiquement d'animaux infectés (Diallo et al., 2007). De nouveaux vaccins, marqués ou basés sur des vecteurs thermostables sont actuellement développés (Diallo et al., 2007 ; Banyard et al., 2010). Pour la valence PPR un des vecteurs expérimentés est le virus vaccinal capripox. Très efficace il a fait la preuve de son pouvoir protecteur à la fois contre la variole caprine et la PPR (Berhe et al., 2003 ; Chaudhary et al., 2009 ; Chen et al., 2010). La voie d'administration orale est également étudiée grâce à des vecteurs à tropisme digestif, elle faciliterait l'administration du vaccin PPR à la faune sauvage (Libeau et al., 2002).

Tableau 1: Facteurs de risque potentiels de la PPR

| | Facteurs de risque | Localisation de l'observation | Références |
|---|---|---|---|
| *Individu* | Espèce | Afrique, Turquie, Inde, Pakistan | Roeder et al., 1994; Diallo, 2000 ; Ozkul et al., 2002 ; Singh et al., 2004 ; FAO, 1999 ; Taylor et al., 2005 ; Lefevre et al., 1990 ; Sow et al., 2008 ; Abraham et al., 2005 ; Awa et al., 2002 ; Abubakar et al., 2011 |
| | Race | Iran, Nigeria, Kazakhstan | Bazarghani et al., 2006; Odo, 2003 ; Lundervold et al., 2004 |
| | Age | Pakistan, Mali, Burkina Faso | Abubakar et al., 2009; Tounkara et al., 1996; Sow et al., 2008 |
| | Sexe | Pakistan | Abubakar et al., 2009 |
| *Climat* | Saison | Pakistan, Inde, Afrique | Abubakar et al., 2009, Singh et al., 2004 ; Taylor,1984 ; Odo, 2003 |
| *Marchés/Commerce/Vols* | Importation de petits ruminants (légale/ illégale) | Arabie Saoudite, Turquie | Al Dubaib, 2009 ; Ozkul et al., 2002, Al Naeem et al., 2000 |
| | Mouvements d'animaux entre pays voisins | Soudan, Turquie, Inde, Chine | Osman et al., 2009; Ozkul et al., 2002 ; Singh et al., 2004 ; Wang et al., 2009. |
| | Marchés d'animaux vivants | Jordanie, Inde, Cameroun | Al Majali et al., 2008 ; Shankar et al., 1988; Martrenchar et al., 1995 |
| | Périodes festives (mouvements d'animaux) | Ghana | Bonniwell, 1980 |
| | Comportements traditionnels et commerciaux | Iran | Bazarghani et al., 2006 |
| | Vols d'animaux | Kenya | Bett et al., 2009 |
| | Partage de points d'eau ou de paturâge | Regions entre 40° de latitude nord et sud | Lefevre et al., 2003 |
| *Systèmes de production* | Pastoralisme et pâturage nomadique<br>Mélange d'animaux locaux et d'animaux transhumants | Pakistan, Inde, Kenya | Abubakar et al, 2009; Bett et al., 2009 ; Singh RP et al, 2004 ; Shankar et al., 1998; Nanda et al., 1996 ; Abubakar et al, 2011 |
| | Grands troupeaux/ forte densité | Jordanie, Turquie, Pakistan | Al Majali et al., 2008; Ozkul et al., 2002; Khan et al., 2008 |
| | Introduction d'animaux achetés au marché | Pakistan, Inde | Abubakar et al., 2009; Singh et al., 2004 ; Abubakar et al., 2011 |
| | Elevage mixte (moutons et chèvres) | Jordanie, Afrique | Al Majali et al., 2008; Anderson and Mc Kay, 1994 |
| | Présence de ruminants sauvages, Chameaux/Dromadaires | Arabie Saoudite, Afrique Centrale et de l'Ouest | Abu Elzein et al., 2004; Couacy-Hymann et al., 2005; Anderson,1995 |
| *Services veterinaires inadéquats* | - Quarantaine inefficace<br>- Accès et disponibilité limités aux soins vétérinaires<br>- Manque de systèmes de surveillance | Iran, Jordanie, Kenya | Bazarghani et al., 2006 ; Al Majali et al., 2008 ; Bett et al, 2009 |

### I.1.4. Epidémiologie théorique : modèles

Des modèles épidémiologiques ont été construits pour d'autres *Morbillivirus* tels que celui de la rougeole (Anderson et May, 1982 ; Fine et al., 1982 ; Keeling, 1997 ; Gay, 2004 ; Papania et al., 2004 ; Keeling et Grenfell, 2002) et celui de la peste bovine (James et Rossiter, 1989 ; Tille et al., 1991 ; Mariner et al., 2005) en vue de l'amélioration de leur contrôle. En ce qui concerne la PPR, la littérature actuelle se limite à une communication scientifique de comparaison de méthodes de modélisation afin de déterminer l'effort optimum à fournir pour son contrôle, que nous redévelopperons par la suite (Roger et al., 2006).

### I.1.5. Impact économique de la maladie

En raison de la confusion probable avec d'autres maladies et du manque d'études spécifiques, l'impact économique de la PPR est très probablement sous-estimé mais il est communément perçu que la PPR est une des contraintes majeures des petits ruminants sous les tropiques (Taylor, 1984 ; Nawathe, 1984).

Dans la plupart des pays où elle est diagnostiquée, la PPR est considérée comme la première maladie des petits ruminants. Néanmoins, et en ne considérant que la situation en Afrique, son incidence économique varie beaucoup des pays côtiers, où elle est très sévère, au Sahel où les épizooties semblent être moins fréquentes. Un rapport publié par Perry et al. et commissionné par le Département pour le Développement International (DFID) du gouvernement du Royaume Uni a identifié la PPR comme une des maladies animales majeures à considérer pour alléger la pauvreté dans les pays où la maladie est endémique (Diallo, 2004 ; Perry et al., 2002).

En 1976, Hamdy et al. ont évalué à près de 1,5 million de dollars américains les pertes annuelles liées à la PPR au Nigéria (Hamdy et al., 1976). Les pertes économiques dues à la PPR seule en Inde ont été estimées à 1800 millions de Roupies soit 39 millions de dollars US (Bandyopadhyay, 2002 ; Gopilo, 2005).

Les impacts économiques de la PPR en Ethiopie n'ont pas été documentés mais elle est considérée comme une des maladies les plus importantes économiquement compte tenu

des taux de morbidité et de mortalité rapportés à travers le pays et de l'importance de la filière petits ruminants (Elzein, 2001 ; Gopilo, 2005 ; Waret-Szkuta et al., 2008).

Au Niger, sur la base d'un taux d'incidence de 14%, Stem a estimé en 1992 qu'un investissement de 2 millions de dollars américains pour la vaccination des chèvres pouvait générer une valeur nette de 24 millions de dollars US au bout de 5 ans (Stem, 1993).

Se fondant sur le principe d'une épizootie sur les chèvres tous les cinq ans, Opasina et Putt (1985) ont estimé qu'une somme annuelle allant de 2,47£ par chèvre dans le pire des cas (fortes pertes) à 0,36£ dans le meilleur des cas (pertes les plus basses) serait profitable dans la prévention efficace de la maladie. En 2001, Elzein fait une étude économique de l'impact de la vaccination PPR avec le vaccin homologue dans les zones East Shewa, North et South Wollo de l'Ethiopie en utilisant un modèle d'arbre de décision. La vaccination contre la PPR est trouvée comme l'alternative optimale. Le retour sur investissement est marginal (1,2-2,5 birr/animal) avec un coût de vaccination de 2,5 birr/animal mais la vaccination était considérée comme justifiée afin d'éliminer un quelconque rôle supposé possible des petits ruminants dans la complication du programme d'éradication de la peste bovine.

Enfin, Awa et al. en 2000 étudient l'impact économique de la vaccination PPR associée à un traitement anthelminthique deux fois par an au Nord Cameroun en comparant les situations avec ou sans prophylaxie. Les bénéfices globaux pour un projet de 5 ans sont estimés à 15 millions de FCFA pour les moutons et 11 millions de FCFA pour les chèvres. Cependant la contribution de la vaccination PPR à ces bénéfices semble minime sans foyer de maladie. Quoiqu'il en soit les auteurs considèrent qu'il est préférable malgré le coût plus important d'appliquer les deux procédures plutôt que de prendre des risques.

Presque tous les échanges de petits ruminants sont effectués entre des pays ayant des zones endémiques PPR alors cette maladie, bien qu'étant sur l'ex-liste A de l'OIE, ne constitue pas une forte contrainte sur les échanges internationaux comme dans le cas de la peste bovine ou de la fièvre aphteuse par exemple (Diallo, 2004). Cependant la surveillance et le contrôle de la maladie restent un enjeu majeur pour ces pays notamment en terme de sécurité alimentaire pour ceux du continent africain.

### I.1.6. Les problématiques associées à la surveillance et au contrôle de la PPR en Afrique

*Problématique 1* : L'occurrence de la PPR est extrêmement variable suivant la zone géographique où elle survient et suivant l'espèce animale concernée. Comprendre les facteurs de risque sur le terrain et la distribution de la maladie est indispensable pour proposer des objectifs réalistes de surveillance de la maladie tant techniquement qu'économiquement. Ces objectifs peuvent être variés et multiples : prévenir l'introduction, protéger ou améliorer certains types de production, contenir la maladie dans une zone, alléger son impact sur la filière petits ruminants, assurer l'export… mais toujours dans un contexte de ressources humaines et financières de plus en plus limitées. Quel type de surveillance à mettre en place pour répondre à quels objectifs et avec quelle pertinence?

*Problématique n°2* : Les modalités du contrôle. La vaccination apparait comme le seul moyen de contrôler la maladie dans les pays en développement au sein d'un contexte de forte voire très forte occurrence de la maladie (Libeau, 2002 ; Diallo, 2004). Comment optimiser ce contrôle en Afrique où le coût des vaccins, de leur administration et la nature des systèmes de production des petits ruminants rendent les campagnes de vaccination difficiles (Banyard et al., 2010) ? Où intervenir et avec quelle intensité ? L'utilisation de la vaccination comme seul moyen de contrôle est-il réaliste ? Y a-t-il un intérêt épidémiologique et socioéconomique au développement de nouveaux vaccins marqués et est-ce réaliste économiquement?

## I.2. OBJECTIFS, PLAN ET RESULTATS ATTENDUS DE LA THESE

### I.2.1. Objectifs

L'objectif principal de la thèse vise à approfondir la compréhension de l'épidémiologie de la PPR afin de tenter d'améliorer les systèmes de surveillance et les mesures de contrôle de la maladie. Les problématiques soulevées peuvent être reformulées sous la forme de deux objectifs secondaires : Comprendre la dynamique et les déterminants de la transmission de

la maladie et identifier les points clés du système hôte/pathogène/environnement sur lesquels potentiellement concentrer les moyens disponibles.

Différentes approches de modélisation sont abordées afin de tenter d'y répondre. Elles sont élaborées à partir de données de terrain majoritairement issues d'Ethiopie, pays de la corne de l'Afrique caractérisé par une forte diversité aussi bien culturelle qu'agro-écologique et la plus forte population d'animaux d'élevage du continent.

La construction d'un modèle déterministe à compartiments (SEIR) a été choisie comme angle d'approche d'une meilleure compréhension du comportement (cycles) de la maladie notamment en terme de transmission, en suivant le principe que les modèles les plus simples sont utilisés pour acquérir une compréhension générale et intuitive des principes clés lorsque les connaissances relatives à l'infection sont limitées ou lorsque l'on étudie une nouvelle question de recherche (Mishra et al., 2011). Par ailleurs, aucun n'avait été construit pour la PPR jusque là. Sans oublier que « tous les modèles sont faux mais certains sont utiles ….» (Box, 1979).

La recherche des données disponibles pour lui donner de la matière a conduit ensuite à se pencher plus précisément sur le cas de l'Ethiopie où une enquête nationale avait été menée en 1999 avant toute campagne massive de vaccination. Ses résultats ont ainsi permis d'identifier certains facteurs de risque de la maladie sur le terrain à l'aide d'un modèle statistique de régression logistique incluant une variable aléatoire pour tenir compte de l'importance spatiale de l'origine des échantillons.

Enfin une enquête de terrain, réalisée à différentes échelles administratives en Ethiopie, sur le suivi des réseaux de partage des points d'eau et des pâturages par les petits ruminants (facteur de risques connus pour la transmission de la maladie) permit d'appréhender l'impact possible de ces structures sur l'organisation d'un programme de surveillance et de contrôle.

### I.2.2. Plan et résultats attendus

Le contexte théorique de la thèse ayant été présenté, nous aborderons dans le deuxième chapitre les outils méthodologiques, c'est-à-dire les modèles qui ont été choisis pour permettre de répondre à nos objectifs. Il s'agit successivement d'un modèle déterministe à

compartiments, d'un modèle statistique de régression logistique et d'un modèle de réseau qui sont chacun rattachés à une application dont les principaux résultats sont résumés.

Etaient attendues dans l'ordre :

- une meilleure compréhension de la dynamique de la transmission de la PPR et la détermination du schéma optimum de vaccination pour son contrôle ;

- la confirmation ou l'infirmation sur le terrain et à l'échelle de l'animal de facteurs de risque décrits dans la littérature, pour ensuite affiner le modèle de transmission

- et enfin la localisation des sites les plus propices à la mise en place de protocoles de surveillance ou de contrôle de la maladie.

Le troisième chapitre regroupe les applications proprement dites c'est-à-dire les articles scientifiques permettant d'avoir le détail de chaque étude ayant été menée. Enfin le quatrième chapitre est une discussion sur la contribution de nos résultats (complétés par les premiers résultats d'une étude du risque d'introduction et de dissémination de la PPR en Afrique qui est en cours) à la surveillance et au contrôle de la PPR basés sur le risque ; il ouvre ensuite des perspectives de recherche.

# II. OUTILS METHODOLOGIQUES : LES MODELES

Un modèle est une simplification de la réalité par définition et tous les modèles sont fondés sur des hypothèses. Le degré de complexité requis est intrinsèquement lié à la question de recherche et limité par les difficultés computationnelles et reste souvent au final une décision subjective (Singer et al., 2011). Il augmente lorsque des informations détaillées sont disponibles, des projections précises sont requises ou de nouvelles hypothèses nécessitent d'être testées (ex : importance de l'hétérogénéité d'un facteur de risque donné). Quoiqu'il en soit les modèles plus complexes ne sont pas nécessairement plus opérationnels que des modèles plus simples car une plus grande incertitude est introduite parallèlement à l'augmentation du nombre de paramètres et d'hypothèses, et les données pour valider les modèles sont rares rendant les résultats difficiles à interpréter (Mishra et al., 2011).

## II.1. Modèles déterministes à compartiments (SEIR)

Dans un contexte où les données sont rares ou difficilement interprétables, représenter la réalité de façon théorique et développer des modèles permet d'approcher les stratégies optimales de contrôle, de prédire le nombre de cas à venir ou de comprendre les schémas d'occurrence d'une maladie. Au niveau du contrôle on peut ainsi étudier et prévoir l'impact d'un élément du programme de santé dans la dynamique de la maladie (McKenzie and Samba, 2004). La théorie des modèles déterministes à compartiments est basée sur la loi d'action de masse, la dynamique d'une épidémie dépendant du taux de contact entre les éléments sains et les éléments infectés, et sur l'hypothèse de panmixage selon laquelle au sein d'un compartiment, la population est homogène et chaque individu a une probabilité similaire d'être infecté (Anderson and May, 1991). Les individus d'une population sont alors représentés comme pouvant être susceptibles (S, sensibles à la maladie), exposés ou latents (E, infectés mais non contagieux), infectieux (I, infectés et contagieux) ou enfin éliminés de la chaine de transmission (R, immunisés ou décédés). La dynamique de l'agent infectieux au

sein de la population peut ainsi être représentée par les équations différentielles ci-dessous (Bailey, 1975) :

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dE}{dt} = \beta SI - \gamma E$$

$$\frac{dI}{dt} = \gamma E - \alpha I$$

$$\frac{dR}{dt} = \alpha I$$

Ainsi la population d'un compartiment passe du statut susceptible au statut infecté au taux β, qui représente le taux de contacts infectieux (nombre de contacts résultant en une infection par unité de temps). La population exposée passe ensuite au statut infectieux au taux γ ($^1/_\gamma$ représentant la durée de la période de latence) et enfin du statut infectieux à celui retiré de la chaine de transmission au taux α ($^1/_\alpha$ représentant la durée de la période infectieuse). Ce système d'équations peut se compliquer pour prendre en compte d'autres compartiments (infectieux mais cliniquement inapparents par exemple) ainsi que des facteurs d'hétérogénéité (espèces, classes d'âge, zones géographiques…). Les transitions d'un stade à l'autre sont déterministes ce qui signifie que, pour les mêmes conditions initiales et les mêmes valeurs de paramètres, les mêmes résultats seront attendus.

Ce type de modèle a été appliqué dans l'article intitulé 'Peste des petits ruminants modelling for disease control decision support' (cf III.1, Article 1) en y introduisant trois strates représentant chacune un sous-groupe d'espèces sensibles (petits ruminants, bovins et buffles). L'objectif était d'approcher le schéma optimum d'intervention dans un contexte endémique en testant différents niveaux de vaccination des petits ruminants.

Les principaux résultats obtenus montrent que la vaccination seule ne semble pas permettre d'envisager l'éradication de la maladie, et si un niveau modéré de vaccination (20%) produit

des résultats encourageant avec une chute importante de l'incidence journalière de la PPR, celle-ci n'est pas beaucoup augmentée lorsque les forts taux de couverture vaccinale préconisés habituellement sur le terrain sont testés. Par ailleurs la situation change radicalement lorsque seule la strate des petits ruminants est considérée, la chute d'incidence apparaissant plus rapidement (un mois post-vaccination au lieu de deux) et étant d'autant plus importante que la couverture vaccinale augmente.

Cependant, si ce modèle permet de donner un cadre d'étude pour une meilleure compréhension de la transmission de la PPR, il sous-tend de nombreuses hypothèses dont celle que tous les animaux dans un sous groupe d'espèce ont la même probabilité d'être infectés et ce de manière indépendante de leur âge, sexe, race, de la nature du virus ou des conditions environnementales locales.

Aussi, ces hypothèses nécessitent d'être investiguées à partir de données de terrain afin d'affiner le modèle en introduisant une, voire deux, stratifications supplémentaires à celle de l'espèce par exemple d'où l'utilisation d'un modèle statistique de régression logistique pour déterminer les facteurs de risque de la maladie.

## II.2. Modèle statistique de régression logistique

On appelle risque, la probabilité d'apparition d'un événement défavorable et facteur de risque ce qui modifie le niveau de ce risque. La présence chez l'individu ou dans son environnement de certaines caractéristiques augmente la possibilité de développer une maladie donnée. La modélisation statistique permet de trouver les facteurs qui caractérisent un groupe de sujets malades par rapport à des sujets sains. Ce sont les variables explicatives du modèle.

La régression logistique modélise les effets de variables explicatives $x_i$ indépendantes sur une variable $y$ résultante binaire qui sera souvent le statut vis-à-vis d'une maladie en épidémiologie ($y$=1 pour des individus/troupeaux etc. qui ont la maladie ; $y$=0 pour des individus/troupeaux etc. qui n'ont pas la maladie). Si π est le risque ou la probabilité de $y$ alors on définit : logodds = log $(\frac{\pi}{1-\pi})$ et on a un modèle général du type :

logodds= $\alpha + \beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_i x_i$ et comme objectif de l'analyse de régression l'estimation des coefficients $\alpha$, $\beta_1, \beta_2, \ldots, \beta_i$

Un exemple de modèle de régression logistique est donné dans l'article intitulé 'Peste des Petits Ruminants (PPR) in Ethiopia : Analysis of a national serological survey' (cf III.2, Article 2) avec comme variables explicatives le sexe, l'âge et l'espèce et comme variable résultante le statut sérologique positif vis-à-vis de la peste des petits ruminants. La variable '*wereda*' que l'on pourrait comparer à une variable 'département' si l'on considérait le territoire français est introduite comme effet aléatoire afin de tenir compte du fait que tous les *wereda* n'ont pas été sélectionnés lors de l'échantillonnage.

L'âge apparait comme le seul facteur associé statistiquement et ce de manière significative au statut séropositif pour la peste des petits ruminants avec un effet linéaire qui suggère que sur le terrain le virus est très immunogène, les animaux infectés le demeurant longtemps.

D'autre part les calculs de séroprévalence (nombre d'échantillons sanguins positifs rapporté au nombre d'individus échantillonnés dans le *wereda*) montrent que le virus de la peste des petits ruminants a circulé quasiment partout en Ethiopie avant que des campagnes de vaccination soient mises en place, l'étude étant antérieure à celles-ci et donc reflétant bien l'infection virale. Cette circulation fut hétérogène, les endroits de faible altitude semblant avoir plus souffert de l'infection que les autres ce qui pourrait s'expliquer par des systèmes de production différents, les échanges et mouvements d'animaux en Ethiopie étant plus fréquents dans les zones de basse altitude et impliquant un plus grand nombre d'animaux.

Enfin la détermination des coefficients de corrélation intra-*kebelle* ($\rho$) (le *kebelle* étant l'échelle administrative juste en dessous de celle du *wereda*) pour chaque *wereda* permet de formuler des hypothèses quant à la circulation ancienne ou récente du virus reflétée par une valeur faible ou élevée de $\rho$ accompagnée d'une valeur faible ou élevée de séroprévalence.

On trouve une corrélation forte entre ρ et le pourcentage d'inhibition[1] qui va dans le sens d'un ρ élevé qui serait le reflet d'une présence épidémique actuelle du virus dans quelques *kebelle* du *wereda* observé.

L'intra-*kebelle* corrélation diminue ensuite avec le temps, se diluant dans le *wereda* suite au remplacement rapide des petits ruminants (3 ans) mais aussi parce que le virus est très immunogène et que les observations sont basées sur des résultats sérologiques qui reflètent le passage du virus avec un léger temps de retard.

Considérant que la PPR est une maladie très contagieuse et que la diffusion dans le *kebelle* mais aussi entre *kebelle* détermine ρ, la faible valeur de ρ dans certains *wereda* pourrait être attribuée à des *wereda* où les animaux de différents *kebelle* se mélangent beaucoup sur les marchés ou au niveau des points d'eau. L'absence d'un schéma spatial évident pour la distribution de ρ à travers l'Ethiopie reflète aussi peut être que la diffusion de la maladie est principalement intervenue à l'intérieur de *wereda* et non pas sous forme de larges épidémies impliquant plusieurs *wereda* contigus.

La distinction mouton/chèvre au sein du groupe des petits ruminants dans le modèle SEIR n'apparait donc pas nécessaire. L'ajout d'une stratification selon l'âge des animaux serait par contre à envisager pour prendre en compte la sensibilité plus importante des jeunes animaux.

Il apparait aussi nécessaire de mieux comprendre la structure des contacts entre les petits ruminants au niveau des marchés mais aussi des points d'eau ou encore des points de pâturages. D'où le choix suivant de la modélisation par la méthode des réseaux sociaux (SNA) qui se prête particulièrement bien à la description de la topologie de la structure des contacts de population d'animaux d'élevage.

[1] Facteur déterminant le statut séropositif de l'animal si il est supérieur à 50 et a priori d'autant plus élevé que l'infection est récente.

## II.3. La modélisation des réseaux sociaux : 'Social Network Analysis' (SNA)

La méthode des réseaux sociaux basée sur la théorie des graphes en mathématiques a été empruntée à la sociologie (Perisse and Nery, 2007). Elle est fondée sur un certain nombre d'éléments et les relations entre ceux-ci. La définition des éléments ou 'nœuds' et des relations qui les relient ou 'liens' dépend entièrement de la question de recherche à laquelle on veut répondre (Ortiz-Pelaez et al., 2006). Les éléments peuvent être des fermes, des animaux, des marchés, des personnes etc. qui établissent des relations avec d'autres éléments. Les définitions de la relation entre les éléments peuvent être multiples : animaux transportés d'une ferme/un lieu/ un marché à un autre, personne/éleveur visitant des fermes, distance entre fermes, partage de lieux communs etc. Ainsi, elle permet la description de la topologie de la structure des contacts de populations animales. L'impact de la structure des réseaux sur les routes potentielles de transmission des maladies infectieuses peut être investigué si les liens entre les nœuds du réseau sont associés à des facteurs de risque connus de transmission de maladie. L'impact de telles structures sur l'efficacité de programmes de surveillance et de contrôle à été montré par plusieurs auteurs (Woolhouse et al., 2005 ; Kiss et al., 2006 ; Kao et al., 2006 ; Kao et al., 2007 ; Ortiz-Pelaez et al., 2006) utilisant les mouvements d'animaux dans des systèmes de production intensive mais peu d'études ont été réalisées dans un contexte où les mouvements d'animaux ne sont pas répertoriés (Van Kerkhove et al., 2009).

Une enquête de terrain fut menée dans un *wereda* des hauts plateaux éthiopiens afin de décrire, d'analyser et de comparer les réseaux de contact générés par le partage par les petits ruminants de points d'eau et de pâturage (cf III.3 Article 3). Les résultats des questionnaires montrent que les modes d'élevage et la composition des cheptels sont similaires à ceux décrits il y a 15 ans. Ils ont permis de représenter les structures de contact aux points d'eau et de paturâge selon la saison (saison des pluies ou saison sèche) et aux échelles respectives du *wereda* et des *kebelle*.

Il apparait que les points de pâturage offrent plus d'opportunités de contact entre animaux appartenant à des *kebelle* ou des villages différents. Ainsi contrairement à l'hypothèse

communément admise que le regroupement des animaux au niveau des points d'eau est un aspect critique de la transmission potentielle d'agents infectieux, il semblerait que des interventions focalisées plutôt sur le partage des points de pâturage seraient plus appropriées dans les hauts plateaux éthiopiens. Ceci est renforcé par le fait que l'on puisse s'attendre à ce que la transmission de maladies soit facilitée aux points de pâturage, les petits ruminants passant plus de temps au pâturage qu'aux points d'eau, ce qui augmente par la même le nombre de troupeaux présents au même endroit au même moment. D'autre part certains villages apparaissent comme à plus faible risque d'introduction de maladies, ne partageant ni point d'eau ni point de pâturage avec les villages voisins peut être à cause de barrières géographiques tels que des montagnes ou des rivières empêchant naturellement les contacts.

# III. APPLICATIONS

### III.1 : Article 1 : Waret-Szkuta A., Collins L.M., Martinez M., Libeau G., Pfeiffer D.U., Roger F. Peste des petits ruminants modelling for disease control decision support. Transboundary and Emerging Diseases, soumis.

### III.2. Article 2 : Waret-Szkuta A., Roger F., Chavernac D., Yigezu L., Libeau G., Pfeiffer D.U., Guitian J. Peste des Petits Ruminants (PPR) in Ethiopia : Analysis of a national serological survey. BMC Veterinary Research 2008, 4 :34

### III.3. Article 3 : A. Waret-Szkuta, A. Ortiz-Pelaez, D.U. Pfeiffer ,F. Roger and F.J. Guitian Herd contact structure based on shared use of water and grazing points in the Highlands of Ethiopia. Epidemiology and Infection 2010, 20: 1-11.

# Peste des petits ruminants modelling for disease control decision support

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Short running title: Peste des petits ruminants modelling

## Summary

Peste des petits ruminants (PPR) is a major disease of domestic and wild small ruminants across Africa and Asia. The development of effective decision-support tools for PPR control needs to include the development of models for defining improved disease control methods. Data from serological surveys in sheep, goats, cattle and buffaloes were used to calculate the forces of infection by means of catalytic models combined with probabilistic functions. Contact parameters were computed from "Who Acquires Infection From Whom" (WAIFW) matrices using probability distributions applied to each input parameter. The resulting R0 distribution had a mean and median of 4.86 and 4.29, respectively. While these values assume a homogeneous host population, the values are very high and it needs to be considered that any successful control effort has to be able to reduce these to below 1 to eliminate the infection. Based on this model, a deterministic compartmental model, stratified by metapopulation and involving stochastic simulation of the contact and transition parameters was implemented to define the levels of vaccination required in small ruminants. A level of 20% was found to decrease the daily infection incidence relative to no control and increasing the vaccine coverage above this level had a relatively small effect when contact with cattle and buffaloes was allowed. This suggests that moderate and achievable levels of vaccination should improve producers' livelihoods but PPR may not be eradicable with vaccination alone and high vaccination targets may not be economically justifiable except in the case where contacts with cattle and wildlife are prevented. This should be interpreted taking into account the assumptions underlying the current model and that the parameter values of this model need to be improved, by conducting targeted field studies.

**Keywords**: Domestic animals; modelling; PPR; vaccination; wildlife

## Introduction

Peste des Petits Ruminants (PPR) is considered to be responsible for mortality and morbidity in cattle and small ruminants in most African countries, the Middle East, the Indian subcontinent and China, with recent outbreaks reported in Tibet in July 2007 (Wang et al., 2009) and in Morocco in July 2008 (Lefevre and Diallo, 1990; Shaila et al., 1996; Diallo, 2003; Gopilo, 2005; Empres, 2008). As a consequence of its high mortality and morbidity and its highly contagious nature, PPR has a significant economic impact in the countries where it occurs (Empres, 2008).

PPR is primarily a disease of goats and sheep. Cattle and pigs are susceptible, but do not normally show symptoms, though clinical signs have been reported in experimentally infected calves (Taylor, 1984). Naturally- and experimentally-induced PPR has also been reported in captive wild ungulates including buffaloes (Furley et al., 1987; Govindarajan et al., 1997), but the role of species other than goats and sheep in the epidemiology of PPR remains unclear. Transmission requires close and direct contact between susceptible and infected animals (Ezeibe et al., 2008). Indirect transmission, although not wholly discounted, seems unlikely considering the low persistence of PPR in the environment (Lefevre and Diallo, 1990). Virus shedding is maximal in the acute stage of the disease and lasts for approximately 14 days unless the infected animal dies before this point (OIE, 2009). Interspecies transmission has been suspected between camels and gazelles (Roger et al., 2001; Abu Elzein et al., 2004) although recent surveys in Sudan tend to suggest this route of dissemination as being unlikely (Khalafalla et al., 2010). Abraham et al. (2005) detected antibody seroprevalence to

70 PPR virus in camels, cattle, goats and sheep in Ethiopia, confirming the occurrence of
71 natural transmission under field conditions.
72 The PPR virus belongs to the family Paramyxoviridae, genus *Morbillivirus,* sharing a
73 genetic and antigenic background with the rinderpest (RP) virus. The resulting possible
74 cross-reaction in serological diagnostic tests and the fact that RP and PPR are clinically
75 indistinguishable in small ruminants has previously led to misclassification potentially
76 favouring the persistence of PPR, especially in India (Shaila et al., 1996), until highly
77 specific tests were developed (Choi et al., 2005). Vaccine cross-neutralization can also
78 occur, therefore heterologous RP vaccines have been widely replaced by homologous
79 ones (OIE, 2009). Indeed, although RP vaccine protects against PPR, the use of RP
80 vaccine in any species is no longer permitted under international standards due to the
81 confusion vaccinal antibodies against RP would cause for surveillance being conducted
82 as part of the validation of global RP eradication (Bastiaensen et al., 2007).
83 Endemic countries suffer epidemic waves of the disease with devastating consequences
84 for small animal production, mortality potentially approaching up to 80% (Empres,
85 2008). Despite effective vaccines and diagnostic tools PPR is still an important disease
86 that continues to spread (Empres, 2009; Banyard et al., 2010). Effective vaccination
87 programmes can help to control the spread of PPR virus, with consequential positive
88 impacts on food security and small farm holder and rural incomes. A better
89 understanding of PPR epidemiology is necessary to design more cost-effective
90 vaccination programmes, and simulation models can have an important role in this
91 process. Epidemiological models have been built for other *Morbillivirus* diseases, such
92 as measles (Anderson and May, 1982; Fine et al., 1982; Keeling, 1997; Gay, 2004,
93 Papania et al., 2004) and RP (James and Rossiter, 1989; Tille et al., 1991; Mariner et al.,
94 2005), however this is the first model to the authors' knowledge on PPR. The objective

of this paper is to develop a deterministic model of PPR dynamics based on inter-species PPR virus transmission between small ruminants, cattle and buffaloes representing the wildlife population. Whilst assuming that PPR virus can only be maintained in small ruminant populations, we assess the impact of different levels of vaccination coverage in small ruminants on PPR daily incidence in an endemic context.

## Materials and methods

### Laboratory technique

Serum samples were analyzed at CIRAD and the National Animal Health Research Center (NAHRC, Sebeta, Ethiopia) using competitive ELISA kits according to manufacturer's instructions (CIRAD and Institute for Animal Health, Pirbright Laboratory, UK respectively). Percentage inhibition (PI) was calculated from optical densities (OD) as:

*PI= 100 – (OD control or test serum/OD monoclonal control)*100*

Samples with PI>50% were considered positive.

These diagnostic tests are reported to have a sensitivity of 94.5% and a specificity of 99.4% relative to the reference test (virus neutralisation test) when used for small ruminants (Libeau, 1995).

### Data Management

Three sets of seroprevalence data were used:

- Serosurveillance data collected on buffaloes and cattle during surveillance for RP under the Pan African Programme for Control of Epizootics (PACE) between 1999 and 2006, tested at CIRAD. Sampling sites were chosen in function of

surveillance needs, disease history, wildlife (including buffaloes) richness, logistics and accessibility. The sampling methods were various: purposive, by immobilisation of individual animals; opportunistic, through cropping or hunting; specific investigation of wildlife disease episodes and mortality or intensive sampling during RP epidemics even if infection was not reported in wildlife (Chardonnet and Kock, 2001).

- Serological results following an outbreak in sheep and goats in Tajikistan in 2004 after purposive sampling. Samples were tested at CIRAD.
- Serological data on sheep and goats collected during the 1999 national serological survey on PPR in Ethiopia using a multistage sampling strategy (Waret-Szkuta et al., 2008).

A total of 4,596 samples with complete information on species, country, animal age in years (<1 ; 1 < 2; 2 < 3; >=3) and sex were used for the two compartmental modelling approaches to be compared, named respectively below catalytic model and transmission model. Age of the animals was estimated by the animal owners. Average age at infection was calculated from the median age of seropositive samples, by species, country and sex. Prior statistical analysis of this dataset showed no statistically significant differences in both models parameters between countries or between sexes. Hence the combined dataset was used to calculate models parameters, rather than repeating the different models for each country involved.

*Population size*

The compartmental models were based on estimates of total population size ($N$) and the population size per species ($N_i$) for an area of 31,415.92 $km^2$ in Kenya. This area

included Meru National Park, where PPR virus can circulate between domestic and wild animals and a 100km surrounding buffer zone. This area was chosen as all buffaloes tested in the surveys considered here originated from the Meru National Park, the field area is well known to the experts consulted at CIRAD, and small ruminant husbandry practices in this area are considered to be similar to those used in other areas of interest, including Ethiopia. Arc View GIS 3.3 for Windows ® was used to define the area based on a map of Kenya (Figure 1).

The minimum, most likely and maximum density of cattle, small ruminants and buffaloes in Kenya (measured as number of heads per square kilometre) were obtained from the Food and Agriculture Organisation of the United Nations (FAO, 2007). These values were then divided by the study area, to calculate the minimum, most likely and maximum densities within the total study area of 31,415.92 $km^2$.

**Catalytic model**

*Next Generation Matrix, R0 and Herd Immunity Threshold*

Animals from one species mix intensively with their own group and less with animals from other species. R0 will therefore be a function of these inter and intragroup contacts. A "Next Generation Matrix" (NGM) (Diekmann et al., 2010) equation was solved to estimate R0 in a heterogeneously mixing population:

$$\begin{pmatrix} R_{srsr} & R_{src} & R_{srb} \\ R_{csr} & R_{cc} & R_{cb} \\ R_{bsr} & R_{cb} & R_{bb} \end{pmatrix} * \begin{pmatrix} x \\ y \\ 1-x-y \end{pmatrix} = R0 * \begin{pmatrix} x \\ y \\ 1-x-y \end{pmatrix}$$

Where $R_{ij} = \beta_{ij} * N_i * Duration\ of\ infectious\ period$,

$R_{ij}$ : number of secondary cases in species *i* resulting from the introduction of one infectious animal from species *j* in a totally susceptible population of *i*;

$\beta_{ij}$ : probability of an individual in *j* effectively contacting a susceptible in *i*;

$N_i$ : population size of species *i*.

*sr*: small ruminants

*c*: cattle

*b*: buffaloes

The herd immunity threshold (*HIT*) was calculated as: $HIT = 1 - 1/R0$.

*Inter- and intra-species effective contact rates ($\beta_i$)*

Animals from one species mix intensively with their own group and less with animals from other species. Due to a lack of data on inter-species interactions, two uniform distributions (*A* and *B*) were used to reflect the uncertainty and variability of the possible contact rates between cattle and buffaloes and cattle and small ruminants. The bounding limits of interaction were fixed based on agreement upon experts opinions obtained at CIRAD on inter-species mixing patterns.

The following matrix equations were solved to calculate the *β*-parameters:

| | *Small ruminants* | *Cattle* | *Buffaloes* | | | | | |
|---|---|---|---|---|---|---|---|---|
| *Small ruminants* | $\beta_{srsr}$ | $\beta_{src}$ | $\beta_{srb}$ | * | $I_{sr}$ | = | $\lambda_{sr}$ |
| *Cattle* | $\beta_{csr}$ | $\beta_{cc}$ | $\beta_{cb}$ | * | $I_c$ | = | $\lambda_c$ |
| *Buffaloes* | $\beta_{bsr}$ | $\beta_{bc}$ | $\beta_{bb}$ | * | $I_b$ | = | $\lambda_b$ |

195

196 *Where* $\beta_{src} = \beta_{csr} = A * \beta_{srsr}$

197 $\beta_{cb} = \beta_{bc} = B * \beta_{cc}$;

198 $\beta_{srb} = \beta_{bsr} = 0$;

199 $A$= Uniform (0.1, 0.5); $B$= Uniform (0, 0.1);

200 $I_{sr}, I_c, I_b$ = Infected number of small ruminants, cattle and buffaloes;

201 $\lambda_{sr}, \lambda_c, \lambda_b$ = Daily force of infection in small ruminants, cattle and buffaloes

202

203 *Force of infection*

204 Force of infection ($\lambda$) was calculated using a catalytic model, based on the average age
205 at infection ($\lambda = 1/a$), according to observed and expected seroprevalence results.
206 Seroprevalence in different age groups was modelled using Poisson distributions in
207 @Risk®, where $\mu$ = number of seropositives observed with an upper limit of the total
208 number of samples in each age-group. Output from the catalytic model was fitted to
209 expected values by minimising deviance. The force of infection was constrained for
210 each species, between the lower limit of the age at which an animal is no longer
211 protected by maternal antibodies and the upper limit of the maximum life expectancy
212 for the species. The model was run until the generated force of infection values
213 stabilized.

214

215 The number of infected animals was calculated based on the number of susceptible
216 animals at endemicity, following the formulae in Anderson and May (1982):

217 Annual force of infection $\lambda = 1/a$ (1)

218 Number susceptible in a type II population $S = (1/(1+L/a)) * N$ (2)

219 Number infected $I = S * \lambda * D$ (3)

220 where $a$ = age at infection, $L$ = average life expectancy, $N$ = population size, and $D$ =
221 average duration of infectiousness.
222
223 The distribution of the number of animals for each species was assumed to be
224 exponential (type II distribution) following the trend of the human population in
225 developing countries.
226
227 *Life expectancy, duration of infectious period*
228 Life expectancy values were based on estimates found in the literature (Meyer, 2009;
229 Anonymous, 2010). In all species groups (small ruminants, cattle, buffaloes), the
230 duration of the infectious period has been estimated by other authors to last between 3.5
231 and 15 days (OIE, 2009).
232
233 *Sensitivity analyses*
234 Sensitivity analyses were carried out using regression analysis and rank correlation
235 calculation in @Risk® to observe the degree to which the uncertainties in the R
236 parameters, HIT and the $\beta$ parameters were affected by the uncertainties of other
237 variables in the model.
238
239 **Transmission model**
240 A deterministic model with four compartments (Susceptible ($S$), Exposed/Infected ($E$),
241 Infectious ($I$), and Resistant ($R$)) was built using the software Powersim Studio 2005 ®
242 (Bailey, 1975), and stratified into three species subgroups: small ruminants (sheep and
243 goats combined) (sr), cattle (c) and buffaloes (b). Model parameter input values are
244 shown in Table 1.

245

246 The following differential equations were used to calculate the transition between the

247 different states of the disease:

248 (1) $\frac{dS_i}{dt} = -\lambda_i S_i(t) - mS_i(t) + (1-v)bN_i$ (Small ruminants)

249 (2) $\frac{dS_i}{dt} = -\lambda_i S_i(t) - mS_i(t) + bN_i$ (Cattle and buffaloes)

250 (3) $\frac{dE_i}{dt} = \lambda_i S_i(t) - fE_i(t) - mE_i(t)$ (All species)

251 (4) $\frac{dI_i}{dt} = fE_i(t) - rI_i(t) - mE_i(t) - m'I_i(t)$ (Small ruminants)

252 (5) $\frac{dI_i}{dt} = fE_i(t) - rI_i(t) - mE_i(t)$ (Cattle and buffaloes)

253 (6) $\frac{dR_i}{dt} = rI_i(t) - mR_i(t) + vbN_i$ (Small ruminants)

254 (7) $\frac{dR_i}{dt} = rI_i(t) - mR_i(t)$ (Cattle and buffaloes)

255

256 Where $v$ = effective vaccination coverage (total vaccine coverage*vaccine efficiency)

257 $b$ = natural birth = $m$ = natural death

258 $\lambda$ = force of infection

259 $m'$ = PPR death

260 $f$ = latency period

261 $r$ = recovery rate

262

263 The model was built using the following assumptions:

264 - All species (buffaloes, cattle, small ruminants) are infectious when infected with

265 PPR.

- Constant population size (birth rate=mortality rate), except for small ruminants that may not survive the infectious period (PPR - related mortality rate).
- Transmission between the different species is determined by their behavioural contact patterns (common grazing for cattle and small ruminants and interface with buffaloes at watering points) and by the probability of effective contact.
- All animals within a species subgroup have equal probability of being infected, independent of age, sex, breed, nature of the virus or local environmental conditions.
- There is heterogeneous mixing between species but homogeneous mixing within species.
- Lifelong immunity (vaccinal or natural).
- Vaccine efficacy ranges between 60-85-95% (A. Diallo, *pers. comm.*).
- Transition between states is constant within species and is independent of host age, environment, sex, or breed.

The initial number of susceptible animals in each species subgroup was calculated as the population size in that subgroup minus the number infected, by assuming an arbitrary, low level starting infection incidence of 200 corresponding to 0.2% of the total population being infected on day 0 – reflecting the level observed in the serology data for each species (sensitivity analyses showed that different starting levels of infection of incidence had no impact on the long-term endemic infection incidence levels, though did have a small effect on long term total population size), and dividing the number of infectious individuals proportionately between the three subgroups according to population size of each group. Daily infection incidence rate per 100,000 animals was calculated as:

((Isr+Ic+Ib) *100,000)/ Total population).

The model was run for 100000 days (275 years), with a start time for vaccination at 31046 days (85 years), once infection incidence had stabilised to a constant level. This time scale allowed us to follow daily infection incidence over a longer period and determine long-term endemicity levels. However, we also provide focal results of the short and medium term around vaccination (day before and up to 10 years after having started to vaccinate) considering all species groups first, and then small ruminants alone.

Five different vaccine coverage levels (0, 20%, 39%, 84%, 95%) were tested for each of three levels of vaccine efficiency (60%, 85% or 95%) to investigate the level of control afforded by the different vaccination programs under the previously calculated minimum, mean and maximum levels of the force of infection.

## Results

The first estimation of R0 using the next generation matrix method gave an estimated mean and median equal to 4.86 and 4.29, respectively, with a best-fit Beta distribution (alpha1=1.1; alpha2=4.5; min=0; max=17.049).

Table 2 summarizes the formulae used to calculate the probabilities of effective contact and the median value of the fitted probability distribution calculated in @Risk®, used as input for the transmission model.

314 The sensitivity analyses showed that for the R parameters, HIT and the $\beta$ parameters,
315 the greatest sensitivities were associated with varying force of infection (correlations
316 varied between 0.55 and 0.97).
317
318 Table 3 shows the day 1, minimum, maximum and day 100,000 infection incidences for
319 each combination of vaccine coverage and efficiency, for each level of force of
320 infection (minimum, mean and maximum force of infection). The graphical
321 representation of the infection incidence of PPR virus in a population where small
322 ruminants, cattle and buffaloes cohabit but only small ruminants are vaccinated is
323 shown in Figure 2 a-d for different vaccination coverage programs. These highlight that
324 for the mean force of infection models, even a small percentage of vaccine coverage
325 results in a significant reduction in day 100,000 infection incidence. The lowest
326 infection incidence is the result of 95% vaccination coverage at 95% efficiency, though
327 the difference between the infection incidence at 20% vaccination coverage and 60%
328 efficiency and that after 95% coverage at 95% efficiency is very small (9.01 and 7.98
329 respectively, in the scenario with minimum force of infection).
330
331 Figure 3 compares the average day 100,000 infection daily incidences for each of the
332 five different coverage levels in each of the different force of infection models. This
333 shows us that for each of the models, having just 20% vaccine coverage will decrease
334 the daily infection incidence relative to no control, and that increasing the vaccine
335 coverage above this level will further reduce the daily infection incidence, but in all
336 models, this reduction is insignificant.
337

Table 4 and Table 5 focus on the short and medium term of the vaccination programs with daily infection incidences given again for each combination of vaccine coverage and efficiency and for each level of force of infection (minimum, mean and maximum force of infection). The main decrease in daily infection incidence occurs the first two months of the programs when small ruminants, cattle and buffaloes cohabit (Table 4). However, when cattle and buffaloes are removed (Table 5) decrease in infection incidence is much quicker (day +30 post-vaccination) and much more important with values tending to zero when the effective vaccine coverage increases.

## Discussion

Modelling is an invaluable tool to better understand the ecology and the epidemiology of an infectious disease and can help decision-making by considering different strategies of vaccination control for example after an outbreak, during an epidemic or in an endemic situation (Mishra et al., 2011). The model presented here is a first theoritical model simulating PPR dynamics.

Although we did not have precise data on the sensitivities and specificities of the diagnostic tests, transmission parameters for cattle and buffaloes and interspecies contact rates, probability distributions were used to describe these parameters to reflect the variability in the possible outcomes each of the parameters can take. Bias associated with the use of expert opinion for interspecies contacts was minimised by the important field experience experts consulted had in and around national parks in Africa. The setting described was that of herd management practices where small ruminants and cattle are reared in free range conditions and where contact with wild animals

362 (especially buffaloes) would be quite rare except during the dry season by the possible
363 sharing of common waterpoints (Tille et al., 1991; Deem et al., 2001).
364
365 In our compartmental model we did not take into account of different levels of
366 susceptibility to PPR virus infection in the animal, population structure and the
367 virulence of the infectious agent when considering transition rates from one status to
368 another. Thus sheep and goats represented as a single group and the higher
369 susceptibility of young animals was not taken into consideration (Waret-Szkuta et al.,
370 2008). Births rates were assumed to be equal to natural mortality rates with additional
371 PPR specific mortality in small ruminants. The population was also assumed closed
372 with no movements into the population from other areas.
373 Our results are quite different from those of other morbillivirus models when
374 considering small ruminants, cattle and buffaloes together (Rossiter and James, 1989;
375 Gay, 2004). According to the results of this model, just 20% vaccine coverage would
376 decrease the infection incidence relative to no control suggesting that moderate and
377 achievable levels of vaccination can have important impact on producers livelihood.
378 However, although logically increasing the vaccine coverage above this level further
379 reduces the infection incidence, it hasalmost no effect. Thus, despite very high level of
380 vaccination (95% coverage at 95% efficiency) the incidence is still of 0.01% per day
381 that could translate into an indefinite persistence in a large susceptible population. Very
382 high levels of vaccination used alone would fail to eradicate the disease. Further,
383 economical justification of high vaccination targets should be sought on a long term
384 scale as investment in vaccination beyond 12% effective coverage does not seem to
385 generate much return in the form of more cases avoided and that the benefits of
386 vaccination seem to be most important during the first two months of the programs.

387 This scenario could be compared to that of preventing contacts of small ruminants with
388 cattle and buffaloes if feasible, as the model outcomes change radically with rates above
389 80% appearing to allow for eradication.
390 Our finding recalls the empirical relationship suggesting that 20% of the population
391 contributes at least 80% of the net transmission potential (Woolhouse et al., 1997). If it
392 would not seem to be an explanation for the result of a homogeneous model, it remains
393 a strong argument for searching out the high-risk sub-populations and eliminating those
394 from the transmission system by vaccination or other means.
395 Further research is needed to collect more data on contact patterns, population structure,
396 differential susceptibility and virulence of the PPR virus (Abubakar et al., 2009).
397 The sensitivity analysis performed for R, HIT and ß parameters shows the highest
398 sensitivities were associated with varying force of infection that was calculated based on
399 the average age at infection. It needs to be noted that this parameter was estimated by
400 the animal owners, and therefore may be imprecise although this should not much affect
401 our parameter estimates.
402
403 In order to estimate R0 a next generation matrix was calculated taking into account that
404 animals mix intensively within subgroups and less between subgroups. The mean R0
405 was 4.86, which is very similar to that produced by the rinderpest lineage 1 model of
406 Mariner et al. 2005. This value of R0 is equivalent to an HIT of 1-1/4.86 or 0.79.
407 However, calculating HIT in such a way is only valid for truly homogeneous
408 populations (Roberts and Heesterbeek, 2003). If vaccination takes place in one of
409 multiple host species then Roberts and Heesterbeek propose the use of the type-
410 reproduction number (T) instead. T numbers are the number of cases of vaccine targeted
411 host per infected case in a susceptible population, either directly or through chains of

412 infection in other host types. This number holds if non-vaccinated hosts sustain an
413 epidemic by themselves. However, in the case of PPR two hosts (sheep and goats)
414 rather than one would be targets for vaccination therefore the T number may be less
415 suitable for estimates of HIT for this disease.
416
417 The modelling exercise suggests that vaccination should allow effective control of PPR
418 virus at the geographic represented here. It needs to be taken into consideration that the
419 parameter values of this model need to be improved, by conducting targeted field
420 studies. However, animal movements into control areas are likely to be a key factor
421 compromising the effectiveness of vaccination campaigns. The importance of this large-
422 scale animal contact and movement patterns should be further investigated using
423 molecular strain typing and specific epidemiological field studies.
424

425 ## Acknowledgements

426 The authors thank Camille Danes, AGIRs unit, CIRAD for creating the map of Kenya.
427 They have no conflict of interest. This study was carried out as part of the EU funded
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429

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577 **Tables**

578 **Table 1:** Description of model input parameters. Where data on separate species was
579 available, this was included in the model. Species code: B= Buffaloes, C=Cattle,
580 Sh=Sheep, G=Goats, SR=small ruminants (sheep and goats combined).

581

582 **Table 2:** Probabilities of effective contact (betas). The medians of the fitted distribution
583 calculated in @Risk for each value were used to calculate the SEIR model input
584 parameters. Betas are a function of the force of infection, the contact patterns and the
585 number of infectious animals, where: "A" – Estimated distribution to explain contact
586 pattern between cattle and goats, Uniform (0.05, 0.3); "B" – Estimated distribution to
587 explain contact pattern between cattle and buffalo, Uniform (0, 0.1); "I" – Number of
588 infectious; "λ"– Force of infection.

589

590 **Table 3:** Starting (day 1), end point (day 100,000) and minimum and maximum
591 infection incidence for all tested combinations of vaccine coverage and efficiency, for
592 minimum, mean and maximum force of infection.

593

594 **Table 4:** Day before starting vaccination (day 31,045) and vaccination days +30, +60,
595 +90, +3,650 infection incidences for all tested combinations of vaccine coverage and
596 efficiency, for minimum, mean and maximum force of infection for all species together.

597

598 **Table 5:** Day before starting vaccination (day 31,045) and vaccination days +30, +60,
599 +90, +3,650 infection incidences for all tested combinations of vaccine coverage and
600 efficiency, for minimum, mean and maximum force of infection for small ruminants
601 alone.

602

603 **Figures**

604 **Figure 1:** Buffer zone (radius of 100kms) around Meru National Park (Kenya). Areas in
605 dark grey are other National Parks.

606

607 **Figure 2:** Infection incidence (per 100,000 heads) for no control (0% vaccine
608 coverage) compared with 39% vaccine coverage (VC), 85% vaccine efficiency (VE) for
609 minimum, mean and maximum force of infection (FOI). (a) 0% VC, minimum FOI; (b)
610 0% VC, mean FOI; (c) 0% VC, maximum FOI; (d) 39% VC, 85%VE, minimum FOI;
611 (e) 39% VC, 85% VE, mean FOI; (f) 39% VC, 85% VE, maximum FOI.

612

613 **Figure 3**: Average infection incidence (per 100,000 heads) at day 100,000 of the
614 simulation for each level of tested vaccine coverage, for minimal, mean and maximal
615 force of infection.

616

617
618

**Table 1**

| Estimated parameters | Units | Value distributions | Model parameters | Units | Calculated model parameters | Model input |
|---|---|---|---|---|---|---|
| Longevity (A) | Years | B: 25<br>C: 10.5<br>SR: 12 | Daily natural birth and death rate (b) and (m) | Heads/Day | 1/A * 1/365 | B: 0.00011<br>C: 0.00026<br>SR: 0.00028 |
| Population (N) | Heads/$km^2$ density land | B: RiskPert(22838.95, 43299.08, 63662.25)<br>C: RiskPert(339506.84, 629376.64, 934389.81)<br>Sh: RiskPert(159344.38, 304416.78, 571705.63)<br>G: RiskPert(317350.63, 550433.90, 890121.94) | Population (N) | Heads/km2 density land | n/a | B: 43270<br>C: 628318<br>SR: 785400 |
| Force of infection (λ) | Years | B: RiskPert(0.041, 0.55, 1.87)<br>C: RiskPert(0.11, 0.59, 1.88)<br>Sh: RiskPert(0.10, 0.57, 1.84)<br>G:RiskPert(0.10, 0.58, 1.85) | Daily force of infection (λ) | Days | λ/365 | **Minimum**<br>B: 0.00011<br>C: 0.0003<br>SR: 0.00028<br>**Mean**<br>B: 0.0015<br>C: 0.0016<br>SR: 0.0016<br>**Maximum**<br>B: 0.0051<br>C: 0.0052<br>SR: 0.0051 |
| Latency period (L) | Days | B: Uniform(3,10)<br>C: Uniform(3, 10)<br>G: RiskPert(3, 5, 10) | Time to infectiousness (f) | Days | 1/L | B: 0.1538<br>C: 0.1538<br>SR: 0.1667 |
| Duration infectious (D) | Days | All species: RiskPert(3.5, 10, 15) | Time to recovery ( r) | Days | 1/D | B: 0.1667<br>C: 0.1667<br>SR: 0.2 |
| PPR specific mortality (m') | % | SR: Uniform (0.5, 0.75) | PPR specific mortality (m') per day | %/days infectious | m'/5 | B: 0<br>C: 0<br>SR: 0.013 |

**Table 2**

| BETAS | Estimated value | Fitted distribution | Median value for model input |
|---|---|---|---|
| ***Bgg*** | λg/(Ig+A Ic) | LogNorm (3.74E-07, 1.63E-07) | 3.41E-07 |
| ***Bcc*** | (λc-A βgg Ig)/(Ic+B Ib) | Gamma (1.76, 3.20E-07) | 4.75E-07 |
| ***Bbb*** | (λb-B βcc Ic)/Ib | LogNorm (3.02E-05, 2.14E-05) | 2.52E-05 |
| ***βgc, βcg*** | A βgg | LogNorm (1.09E-07, 6.08E-08) | 9.57E-08 |
| ***βgb, βbg*** | 0 | 0 | 0 |
| ***βcb, βbc*** | B βcc | Beta (2.76E-08, 2.90E-08) | 1.95E-08 |

**Table 3**

| Force of infection | Vaccine coverage (%) | Vaccine efficiency (%) | Effective vaccine coverage (%) | Infection incidence (100,000 heads) | | | |
|---|---|---|---|---|---|---|---|
| | | | | Day 1 | Minimum | Maximum | Day 100,000 |
| **Minimum** | 0 | n/a | 0 | 199.96 | 94.99 | 818.48 | 95.29 |
| | 20 | 60 | 12 | 199.96 | 9.01 | 818.48 | 9.01 |
| | 20 | 85 | 17 | 199.96 | 8.62 | 818.48 | 8.62 |
| | 20 | 95 | 19 | 199.96 | 8.52 | 818.48 | 8.52 |
| | 39 | 60 | 23.4 | 199.96 | 8.37 | 818.48 | 8.37 |
| | 39 | 85 | 33.15 | 199.96 | 8.18 | 818.48 | 8.18 |
| | 39 | 95 | 37.05 | 199.96 | 8.14 | 818.48 | 8.14 |
| | 84 | 60 | 50.4 | 199.96 | 8.05 | 818.48 | 8.05 |
| | 84 | 85 | 71.4 | 199.96 | 7.99 | 818.48 | 7.99 |
| | 84 | 95 | 79.8 | 199.96 | 7.98 | 818.48 | 7.99 |
| | 95 | 60 | 57 | 199.96 | 8.02 | 818.48 | 8.02 |
| | 95 | 85 | 80.75 | 199.96 | 7.98 | 818.48 | 7.99 |
| | 95 | 95 | 90.25 | 199.96 | 7.98 | 818.48 | 7.98 |
| **Mean** | 0 | n/a | 0 | 199.96 | 96.31 | 199.96 | 108.35 |
| | 20 | 60 | 12 | 199.96 | 70.65 | 199.96 | 70.65 |
| | 20 | 85 | 17 | 199.96 | 70.60 | 199.96 | 70.60 |
| | 20 | 95 | 19 | 199.96 | 70.59 | 199.96 | 70.59 |
| | 39 | 60 | 23.4 | 199.96 | 70.57 | 199.96 | 70.57 |
| | 39 | 85 | 33.15 | 199.96 | 70.56 | 199.96 | 70.56 |
| | 39 | 95 | 37.05 | 199.96 | 70.56 | 199.96 | 70.56 |
| | 84 | 60 | 50.4 | 199.96 | 70.56 | 199.96 | 70.56 |
| | 84 | 85 | 71.4 | 199.96 | 70.58 | 199.96 | 70.58 |
| | 84 | 95 | 79.8 | 199.96 | 70.58 | 199.96 | 70.58 |
| | 95 | 60 | 57 | 199.96 | 70.56 | 199.96 | 70.56 |
| | 95 | 85 | 80.75 | 199.96 | 70.58 | 199.96 | 70.58 |
| | 95 | 95 | 90.25 | 199.96 | 70.59 | 199.96 | 70.59 |
| **Maximum** | 0 | n/a | 0 | 199.96 | 168.02 | 3118.16 | 207.67 |
| | 20 | 60 | 12 | 199.96 | 139.18 | 3118.16 | 140.73 |
| | 20 | 85 | 17 | 199.96 | 138.66 | 3118.16 | 139.60 |
| | 20 | 95 | 19 | 199.96 | 138.53 | 3118.16 | 139.31 |
| | 39 | 60 | 23.4 | 199.96 | 138.32 | 3118.16 | 138.86 |
| | 39 | 85 | 33.15 | 199.96 | 138.07 | 3118.16 | 138.30 |
| | 39 | 95 | 37.05 | 199.96 | 138.00 | 3118.16 | 138.17 |
| | 84 | 60 | 50.4 | 199.96 | 137.87 | 3118.16 | 137.90 |
| | 84 | 85 | 71.4 | 199.96 | 137.76 | 3118.16 | 137.76 |
| | 84 | 95 | 79.8 | 199.96 | 137.74 | 3118.16 | 137.74 |
| | 95 | 60 | 57 | 199.96 | 137.83 | 3118.16 | 137.84 |
| | 95 | 85 | 80.75 | 199.96 | 137.74 | 3118.16 | 137.74 |
| | 95 | 95 | 90.25 | 199.96 | 137.74 | 3118.16 | 137.74 |

**Table 4**

| Force of infection | Vaccine coverage (%) | Vaccine efficiency (%) | Infection incidence (100,000 heads) Day 31045 (-1 day vaccination starts (dvs)) | Day 31076 (+30 dvs) | Day 31106 (+60 dvs) | Day 31136 (+90 dvs) | Day 34696 (+3650 dvs) |
|---|---|---|---|---|---|---|---|
| **Mini-mum** | 0 | n/a | 96.588 | 96.583 | 96.579 | 96.574 | 96.111 |
| | 20 | 60 | 96.588 | 23.778 | 14.769 | 14.519 | 13.011 |
| | 20 | 85 | 96.588 | 19.250 | 14.226 | 14.165 | 13.368 |
| | 20 | 95 | 96.588 | 17.202 | 14.113 | 14.077 | 12.566 |
| | 39 | 60 | 96.588 | 17.047 | 13.971 | 13.940 | 12.426 |
| | 39 | 85 | 96.588 | 15.908 | 13.793 | 13.768 | 12.252 |
| | 39 | 95 | 96.588 | 15.129 | 13.748 | 13.727 | 12.210 |
| | 84 | 60 | 96.588 | 15.299 | 13.664 | 13.641 | 12.123 |
| | 84 | 85 | 96.588 | 15.052 | 13.607 | 13.586 | 12.068 |
| | 84 | 95 | 96.588 | 15.000 | 13.598 | 13.576 | 12.058 |
| | 95 | 60 | 96.588 | 15.194 | 13.639 | 13.617 | 12.099 |
| | 95 | 85 | 96.588 | 14.996 | 13.597 | 13.575 | 12.057 |
| | 95 | 95 | 96.588 | 14.556 | 13.589 | 13.570 | 12.052 |
| **Mean** | 0 | n/a | 98.754 | 98.758 | 98.762 | 98.766 | 99.276 |
| | 20 | 60 | 98.754 | 74.705 | 70.786 | 70.702 | 70.697 |
| | 20 | 85 | 98.754 | 72.408 | 70.689 | 70.666 | 70.662 |
| | 20 | 95 | 98.754 | 72.101 | 70.668 | 70.657 | 70.653 |
| | 39 | 60 | 98.754 | 71.698 | 70.650 | 70.644 | 70.640 |
| | 39 | 85 | 98.754 | 71.637 | 70.634 | 70.628 | 70.623 |
| | 39 | 95 | 98.754 | 71.090 | 70.626 | 70.623 | 70.619 |
| | 84 | 60 | 98.754 | 71.168 | 70.618 | 70.616 | 70.612 |
| | 84 | 85 | 98.754 | 71.098 | 70.614 | 70.610 | 70.608 |
| | 84 | 95 | 98.754 | 71.082 | 70.612 | 70.609 | 70.607 |
| | 95 | 60 | 98.754 | 71.138 | 70.616 | 70.613 | 70.610 |
| | 95 | 85 | 98.754 | 71.276 | 70.613 | 70.609 | 70.607 |
| | 95 | 95 | 98.754 | 70.933 | 70.610 | 70.608 | 70.607 |
| **Maxi-mum** | 0 | n/a | 180.988 | 181.002 | 181.015 | 181.028 | 182.185 |
| | 20 | 60 | 180.988 | 143.690 | 139.290 | 139.185 | 139.262 |
| | 20 | 85 | 180.988 | 141.188 | 138.687 | 138.665 | 138.713 |
| | 20 | 95 | 180.988 | 140.085 | 138.542 | 138.532 | 138.572 |
| | 39 | 60 | 180.988 | 139.890 | 138.333 | 138.324 | 138.352 |
| | 39 | 85 | 180.988 | 139.153 | 138.070 | 138.065 | 138.077 |
| | 39 | 95 | 180.988 | 138.710 | 138.004 | 138.001 | 138.009 |
| | 84 | 60 | 180.988 | 138.716 | 137.875 | 137.871 | 137.872 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 84 | 85 | 180.988 | 138.533 | 137.789 | 137.786 | 137.784 |
| 84 | 95 | 180.988 | 138.494 | 137.774 | 137.771 | 137.770 |
| 95 | 60 | 180.988 | 138.638 | 137.837 | 137.833 | 137.833 |
| 95 | 85 | 180.988 | 138.494 | 137.773 | 137.770 | 137.768 |
| 95 | 95 | 180.988 | 138.258 | 137.762 | 137.760 | 137.759 |

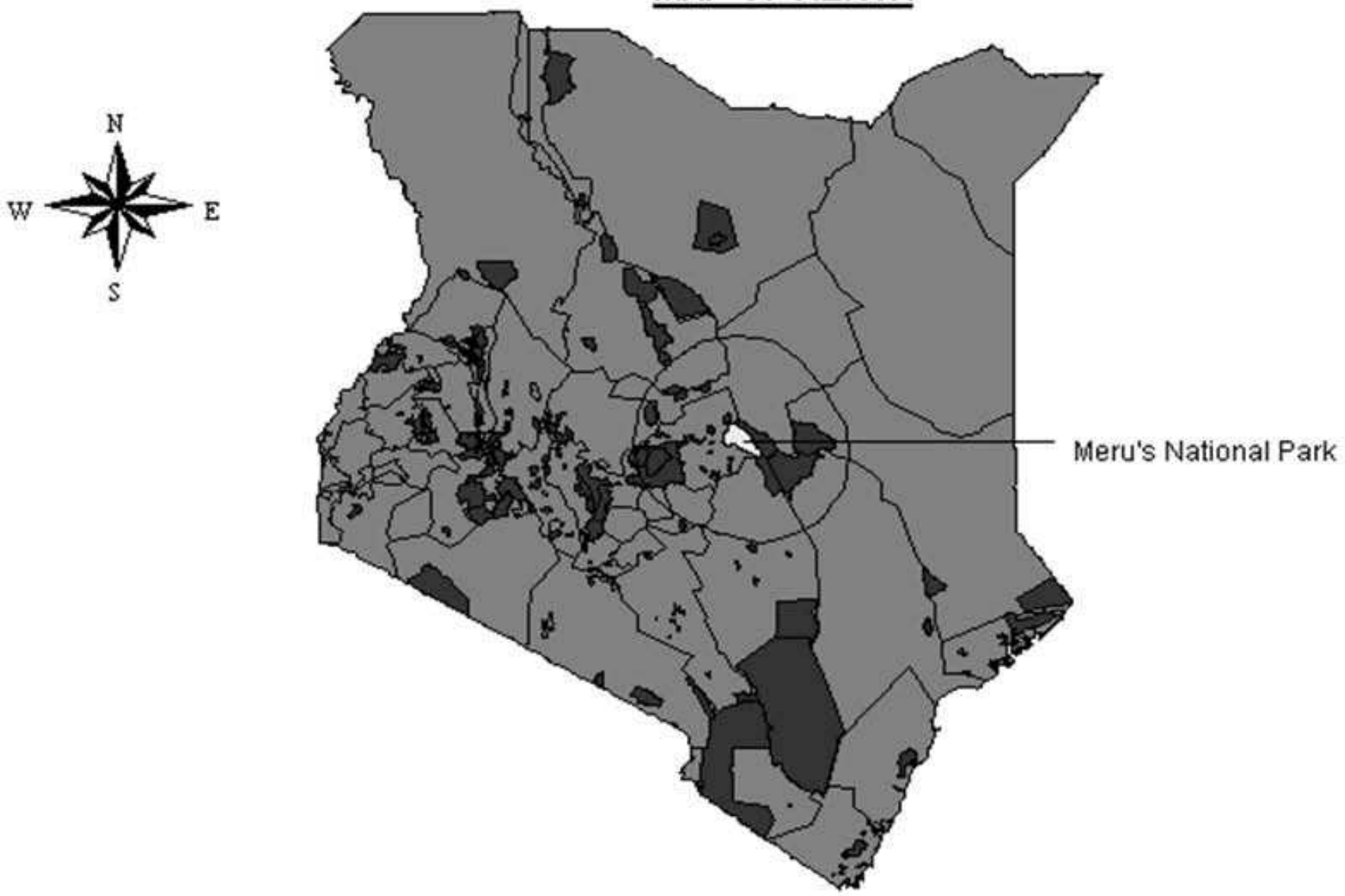
**Table 5**

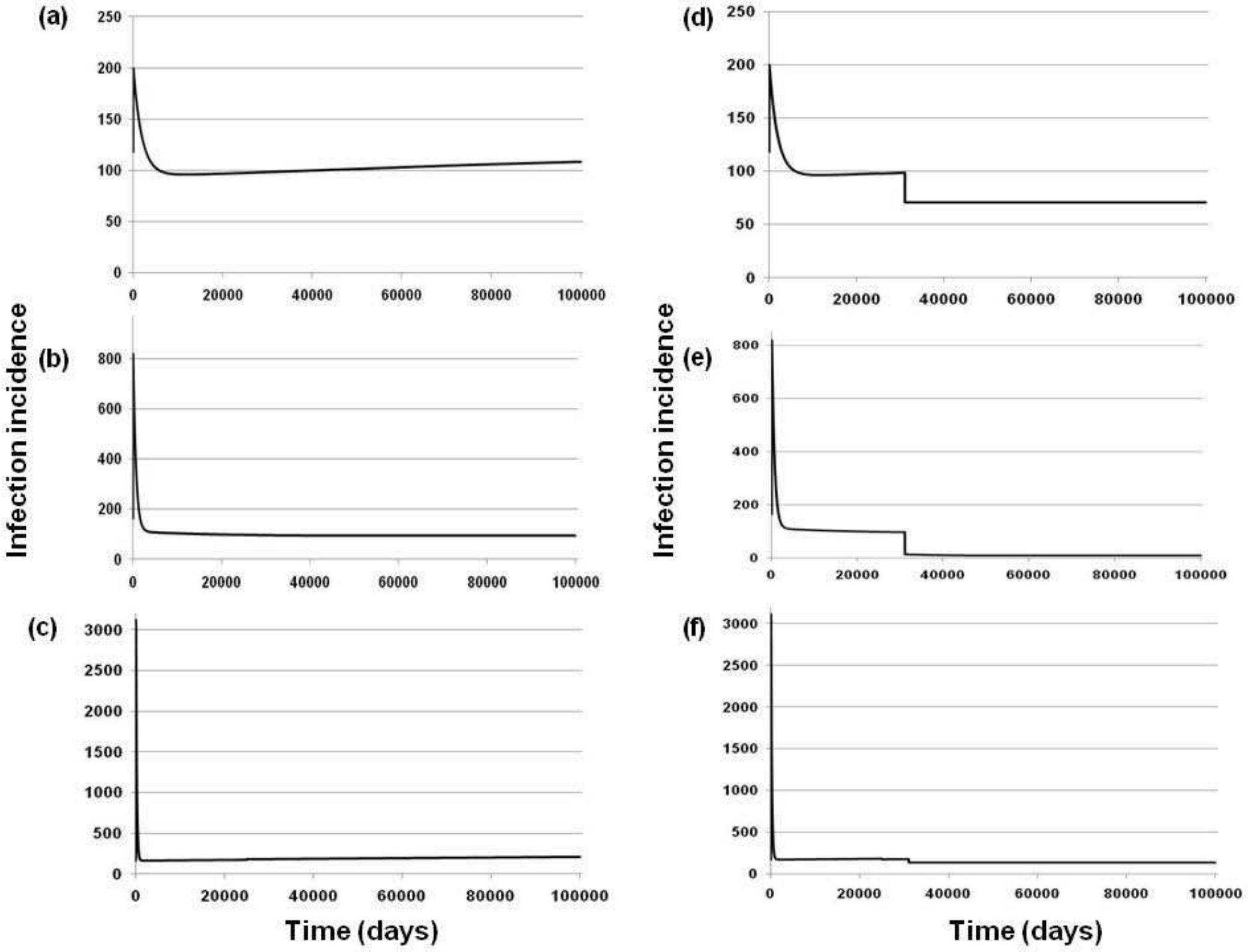
| | | | Infection incidence (100,000 heads) | | | | |
|---|---|---|---|---|---|---|---|
| **Force of infection** | **Vaccine coverage (%)** | **Vaccine efficiency (%)** | **Day 31045 (-1 day vaccination starts (dvs))** | **Day 31076 (+30 dvs)** | **Day 31106 (+60 dvs)** | **Day 31136 (+90 dvs)** | **Day 34696 (+3650 dvs)** |
| **Minimum** | 0 | n/a | 59.293 | 59.293 | 59.293 | 59.293 | 59.293 |
| | 20 | 60 | 59.293 | 6.949 | 0.370 | 0.193 | 0.189 |
| | 20 | 85 | 59.293 | 3.788 | 0.153 | 0.119 | 0.119 |
| | 20 | 95 | 59.293 | 2.344 | 0.117 | 0.101 | 0.101 |
| | 39 | 60 | 59.293 | 2.292 | 0.086 | 0.074 | 0.074 |
| | 39 | 85 | 59.293 | 1.553 | 0.047 | 0.040 | 0.040 |
| | 39 | 95 | 59.293 | 1.015 | 0.036 | 0.031 | 0.031 |
| | 84 | 60 | 59.293 | 1.179 | 0.020 | 0.014 | 0.014 |
| | 84 | 85 | 59.293 | 1.030 | 0.008 | 0.003 | 0.003 |
| | 84 | 95 | 59.293 | 0.998 | 0.006 | 0.002 | 0.002 |
| | 95 | 60 | 59.293 | 1.115 | 0.015 | 0.010 | 0.010 |
| | 95 | 85 | 59.293 | 0.995 | 0.006 | 0.001 | 0.001 |
| | 95 | 95 | 59.293 | 0.683 | 0.003 | 0.000 | 0.000 |
| **Mean** | 0 | n/a | 93.899 | 93.899 | 93.899 | 93.899 | 93.899 |
| | 20 | 60 | 93.899 | 11.517 | 1.340 | 1.074 | 1.068 |
| | 20 | 85 | 93.899 | 6.394 | 0.726 | 0.674 | 0.674 |
| | 20 | 95 | 93.899 | 4.078 | 0.599 | 0.575 | 0.575 |
| | 39 | 60 | 93.899 | 3.901 | 0.437 | 0.418 | 0.418 |
| | 39 | 85 | 93.899 | 2.613 | 0.237 | 0.225 | 0.225 |
| | 39 | 95 | 93.899 | 1.732 | 0.186 | 0.179 | 0.179 |
| | 84 | 60 | 93.899 | 1.924 | 0.090 | 0.082 | 0.082 |
| | 84 | 85 | 93.899 | 1.644 | 0.027 | 0.019 | 0.019 |
| | 84 | 95 | 93.899 | 1.586 | 0.016 | 0.009 | 0.009 |
| | 95 | 60 | 93.899 | 1.805 | 0.063 | 0.054 | 0.054 |
| | 95 | 85 | 93.899 | 1.581 | 0.015 | 0.008 | 0.008 |
| | 95 | 95 | 93.899 | 1.083 | 0.007 | 0.002 | 0.002 |
| **Maximum** | 0 | n/a | 102.451 | 102.451 | 102.451 | 102.451 | 102.451 |
| | 20 | 60 | 102.451 | 14.043 | 3.613 | 3.362 | 3.357 |
| | 20 | 85 | 102.451 | 8.117 | 2.187 | 2.135 | 2.134 |
| | 20 | 95 | 102.451 | 5.507 | 1.849 | 1.825 | 1.825 |
| | 39 | 60 | 102.451 | 5.044 | 1.352 | 1.332 | 1.332 |
| | 39 | 85 | 102.451 | 3.300 | 0.733 | 0.721 | 0.721 |
| | 39 | 95 | 102.451 | 2.254 | 0.581 | 0.573 | 0.573 |
| | 84 | 60 | 102.451 | 2.266 | 0.272 | 0.262 | 0.262 |
| | 84 | 85 | 102.451 | 1.834 | 0.070 | 0.062 | 0.062 |

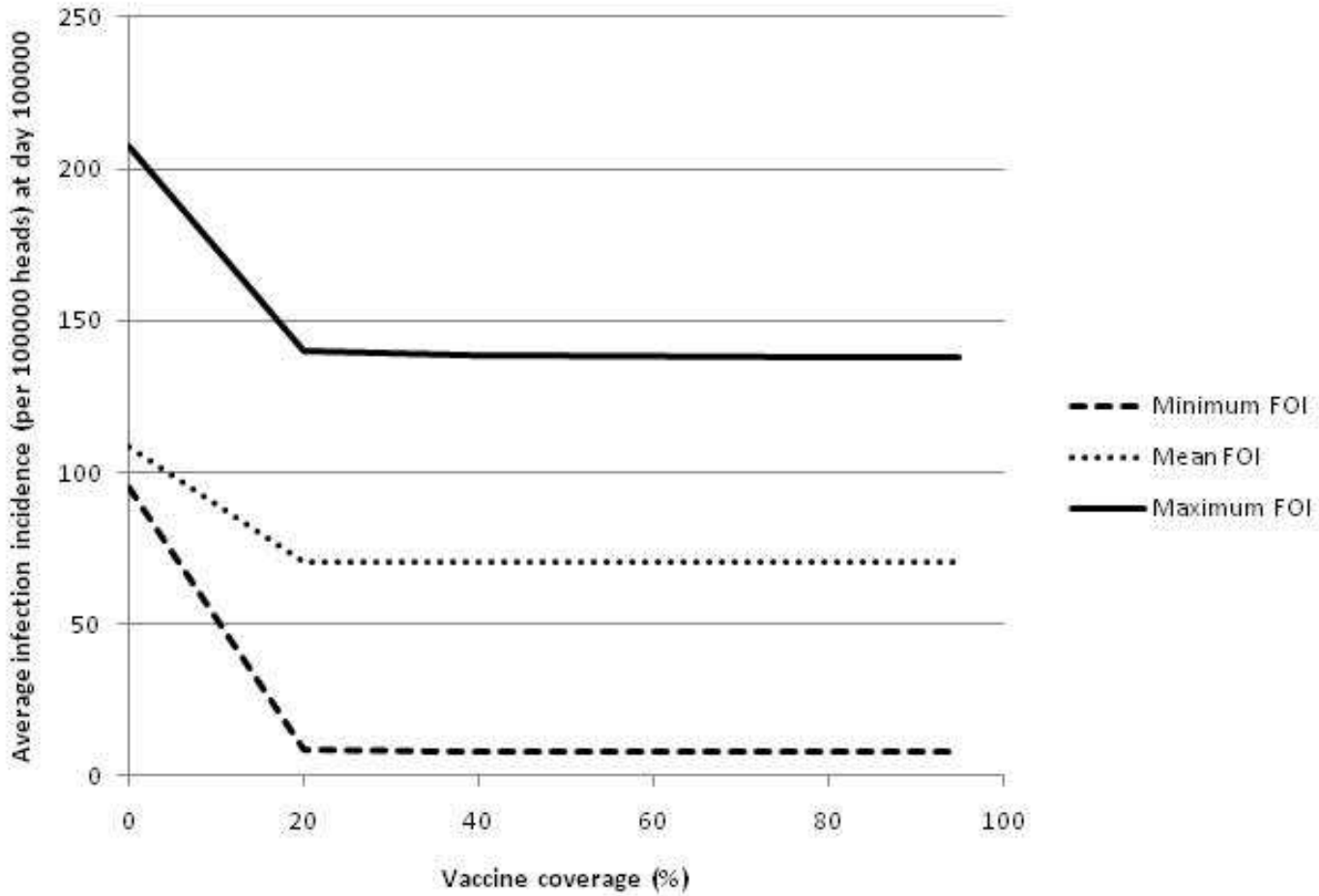
| | | | | | | |
|---|---|---|---|---|---|---|
| 84 | 95 | 102.451 | 1.749 | 0.036 | 0.028 | 0.028 |
| 95 | 60 | 102.451 | 2.081 | 0.184 | 0.175 | 0.175 |
| 95 | 85 | 102.451 | 1.741 | 0.033 | 0.025 | 0.025 |
| 95 | 95 | 102.451 | 1.186 | 0.011 | 0.006 | 0.006 |

500 0 500 1000 Kilometers

# MAP OF KENYA




(a)
(b)
(c)
(d)
(e)
(f)
Infection incidence
Infection incidence
Time (days)
Time (days)
0
20000
40000
60000
80000
100000


250
200
150
100
50
0
Average infection incidence (per 100000 heads) at day 100000
0
20
40
60
80
100
Vaccine coverage (%)
Minimum FOI
Mean FOI
Maximum FOI

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# Peste des Petits Ruminants (PPR) in Ethiopia: Analysis of a national serological survey

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Published: 12 September 2008

*BMC Veterinary Research* 2008, **4**:34 doi:10.1186/1746-6148-4-34

Received: 22 April 2008
Accepted: 12 September 2008

This article is available from: http://www.biomedcentral.com/1746-6148/4/34



## Abstract

**Background:** Peste des petits ruminants (PPR) is a contagious viral disease of small ruminants in Africa and Asia. In 1999, probably the largest survey on PPR ever conducted in Africa was initiated in Ethiopia where 13 651 serum samples from 7 out of the 11 regions were collected and analyzed by competitive enzyme-linked immunosorbent assay (cELISA). The objective of this paper is to present the results of this survey and discuss their practical implications for PPR-endemic regions.

**Methods:** We explored the spatial distribution of PPR in Ethiopia and we investigated risk factors for positive serological status. Intracluster correlation coefficients (ρ), were calculated for 43 *wereda* (administrative units).

**Results:** Seroprevalence was very heterogeneous across regions and even more across *wereda*, with prevalence estimates ranging from 0% to 52.5%. Two groups of *weredas* could be distinguished on the basis of the estimated ρ: a group with very low ρ ($\rho < 0.12$) and a group with very high ρ ($\rho > 0.37$).

**Conclusion:** The results indicate that PPRV circulation has been very heterogeneous, the values for the ρ may reflect the endemic or epidemic presence of the virus or the various degrees of mixing of animals in the different areas and production systems. Age appears as a risk factor for seropositive status, the linear effect seeming to confirm in the field that PPRV is highly immunogenic. Our estimates of intracluster correlation may prove useful in the design of serosurveys in other countries where PPR is of importance.

## Background

Peste des Petits Ruminants (PPR) is a severe and highly infectious viral disease of small ruminants. The PPR virus (PPRV) belongs to the genus *Morbillivirus* in the family Paramyxoviridae. It is closely related to the rinderpest virus of bovines and buffaloes, distemper virus of dogs and other wild carnivores, human measles virus and Morbilliviruses of marine mammals [1-4]. In small ruminants, infection by PPRV is characterized by sudden depression, fever, nasal and ocular discharge, diarrhoea and occasion-

ally death. Morbidity in the range of 10 to 80% and mortality proportions from 0 to 90% have been reported. The wide range of reported values is likely to be influenced by differences between species (sheep or goats), production systems and levels of natural or acquired immunity [5-10].

PPR was first described in West Africa in 1942 [11]. Nowadays the disease is recognized as responsible for mortality and morbidity across most of the sub-Saharan African countries situated north of the equator, in the Arabian Peninsula, in India and in numerous other countries in Asia [6,12-14]. Although nationwide serosurveys have been conducted in countries such as the sultanate of Oman, Turkey, Jordan and India, information on the frequency and distribution of PPR is often lacking when control or eradication campaigns are initiated [15-18]. Control of PPR in endemic areas relies mainly in vaccination [19,20]. In 1989 a homologous vaccine that induces lifelong immunity in both sheep and goats was developed [6,21-23]. The vaccine is innocuous on pregnant sheep and goats at any stage of gestation and induces the production of colostral anti-PPR antibodies that have been found in kids up to 3 months old [6,23].

Ethiopia has the most important livestock population in Africa and is ranked 9$^{th}$ in the world [24]. The livestock sub sector accounts for 40% of the agricultural gross domestic product (GDP) and 20% of the total GDP (Aklilu Y. An audit of livestock marketing status in Kenya, Ethiopia and Sudan. PACE/OUA/IBAR, 2002) without considering the livestock contribution in terms of traction power, fertilizing and mean of transport. Sheep and goat populations are estimated to be 20.7 million and 16.4 million respectively [25]. Sheep and goats contribute 25% of the meat domestically consumed with a production surplus mainly being exported as live animals [26,27]. Both species also contribute 50% of the domestic needs in wool, about 40% of skins and 92% of the value of hides and skin exported [28]. The annual production of sheep and goat meat is estimated as 56 560 and 28 650 tonnes respectively [24]. PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat herd in the Afar region, East of the country [9,29]. Clinical and serological evidence of its presence has been reported by Taylor (1984) and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa [29]. During the nineties, several small serological surveys were conducted, mainly east of an imaginary line that would run parallel to the Rift valley and pass through Addis Ababa. In 1994 Roger and Bereket (CIRAD-EMVT report n°96006, Montpellier, 1996) found seroprevalences of up to 33% in sheep and 67% in goats near selected urban areas. In 1996 Gelagay found that 14.6% of sheep sampled along 4 roads from Debre Berhan to Addis Ababa were seropositive [30]. In 1997 Yayerade found up to 100% of seropositive individuals in groups of adult male sheep and animals that survived suspected outbreaks. Although these studies provide very limited and potentially biased information about the frequency and distribution of PPR in Ethiopia, they clearly suggest that the virus has been circulating extensively among the small ruminant population of Ethiopia during the nineties. Based on the reported morbidity and mortality of the infection and the size and structure of the small ruminant sector it is likely that PPR became one of the most economically important livestock diseases in the country [12,31].

In 1999, a serological survey on PPR was conducted in Ethiopia with the aim of informing a subsequent vaccination campaign which would be the first large scale vaccination campaign against PPR in the country. As part of the survey, a total of 13 753 sheep and goats were sampled. To our knowledge, this is the largest serological survey on PPR ever conducted in Africa. The objective of this paper is to describe the results of this survey and discuss its practical implications.

## Methods

### *Administrative structure and distribution of small ruminants in Ethiopia*

The Ethiopian administrative structure has frequently been subject to modification. To date there are 11 Regions or States or *Kelel* composed of 71 zones. These zones include about 546 districts or *wereda* or *woreda*. Each *wereda* is composed of *kebelles* or *Peasant Associations* that are an aggregation of *got*, a *got* being a group of 3 to 5 villages although the difference between *got* and village is sometimes unclear in the field [25]. The very diverse relief of the country determines several geoclimatic zones. The central part is characterized by mountainous massifs and covers half of the territory. It is a zone of Highlands ranging from 2 300 to 3 500 m called *Dega* surrounded by a temperate transition zone between 1 500 and 2 300 m called *Woinadega* that dives in the central Rift Valley towards the south west. East the tectonic deflection opens on the lowland areas *Bereha* and *Kola* (0 to 1 200 m), zones of pastoral nomadic livestock husbandry [32].

In 1995 about three quarters of the sheep stock was located in the Ethiopian highlands (> 1 500 m) [33]. A recent report (Aklilu Y. consultancy report USAID/Ethiopia and EGAT Office of USAID/Washington, 2005) suggests that nowadays around half of the sheep are kept in the highlands and half in the lowlands.

### *The 1999 Survey*

According to a report of the Ethiopian Ministry of Agriculture and Rural Development of 2005 the serological sur-

vey was initiated in 1999 to determine the distribution of PPR across the country and to identify areas of increased risk. Compared to the previous studies the geographical coverage is extended to north and western parts of the country. The objective of the survey was to inform the design of a strategy for cost effective control of the disease. The survey was implemented as a subcomponent of the animal health component under the National Livestock Development Project (NLDP) of the Ethiopian Ministry of Agriculture. This project started in 1999 and was financed by the African Development Bank (ADB). The original plan was to collect 8 000–12 000 sera samples through 7 regional veterinary laboratories located in 7 regional states to inform a 3 year vaccination campaign to be started in 2004. Thus vaccination would be implemented in those *wereda* identified by the regional states as endemic for PPR as well as in the neighbouring *wereda*.

Multistage sampling was the chosen sampling strategy, with 4 hierarchical stages as illustrated in Table 1. The first level of selection was the region; only those regions with a veterinary laboratory (7 out of the 11 regions) were selected. Within each of the selected regions (Afar, Amhara, Benishangul Gumuz, Oromia, SNNPR, Somali, Tigray) *weredas*, *kebelles* and villages were randomly selected (Epidemiology unit, Ministry of Agriculture and Rural Development personal communication). Within each of the selected villages, 20 animals (either sheep or goats) were supposed to be randomly selected but were most probably purposively selected because of field and time constraints although we have not been able to completely ascertain the details of the selection process (Laikemariam Yigezu, former PACE coordinator and head of Microbiology unit at the Sebeta laboratory, personal communication).

### *Laboratory techniques*

Serum samples were analyzed by the National Animal Health Research Center (NAHRC, Sebeta, Ethiopia) using a competitive ELISA kit according to the instructions of the manufacturer (Institute for Animal Health, Pirbright Laboratory, UK). The ELISA micro-plates were read with an immunoskan reader (Flow laboratories, UK) with an inference filter of 492 nm. The reader was connected to a computer loaded with ELISA Data Information (EDI) software (FAO/IAEA, Vienna, Austria), which was used to automate the reading and calculation of the percentage of inhibition (PI) values. The OD (Optical Density) values were converted to percentage inhibition using the following formula:

$$PI = 100 - (OD\ control\ or\ test\ serum / OD\ monoclonal\ control) * 100$$

The samples with PI > 50% (cut-off) were considered as positives.

### *Data management*

The data were entered and stored electronically in Microsoft Office Access 2003. The fields included in the database are presented in Table 2. Laboratory results and field information collected during the sampling were entered into the database. When age was given as a binary variable (young vs. adult; n = 157 entries), it was considered to be a missing value. When age was given as number of months or years of age (values ranging from 6 months to 10 years old; n = 465) the original variable was recategorized into 4 categories (< 1; 1 < 2; 2 < 3; > = 3) to match with the categories given for the remaining 4 181 animals for which age was given using these four categories.

### *Data analysis and spatial description*

Descriptive statistics of the studied variables were obtained. Within each *wereda*, the following parameters were obtained:

- seroprevalence (number of positive valid samples/ number of individuals sampled in the *wereda*)

- intra-*kebelle* correlation coefficient (ρ) for the 43 *weredas* for which information about the *kebelle* of origin of the samples was available calculated as:

**Table 1: Structure of the different administrative levels of sampling**

| Included in the above administrative level | Region (Ref) | *wereda* | *kebelle* | village |
|---|---|---|---|---|
| Total | 7 | 84 | | |
| Mode | | 9 | 5 | 5 |
| Average | | 12.14 | 4.98* | 4.45** |
| Range | | 8 – 19 | 2 – 9 | 1 – 5 |

*average number of *kebelle* per *wereda* where *kebelle* level was available

**average number of villages per *kebelle* where *village* level was available

The table shows the 4 administrative levels of sampling in the 1999 national serological survey on PPR in Ethiopia. The first level of selection was the region with 7 regions selected. Within each of the regions *weredas*, *kebelles* and villages were randomly selected. For each level the mode, average and range of units included in the above administrative level are presented.

**Table 2: List of relevant variables included in the database along with the number of observations available**

| Variable | Number of samples for which it was recorded | % of samples for which it was recorded |
|---|---|---|
| **Localisation** | | |
| Region | 13 651 | 100 |
| *Wereda* | 13 613 | 99.7 |
| *Kebelle* | 9 328 | 68.3 |
| Village | 980 | 7.2 |
| **Species**[a] | 13 651 | 100 |
| **Age**[a] | 4 648 | 34 |
| **Sex**[a] | 5 868 | 43 |
| **Results** | | |
| OD | 13 651 | 100 |
| PI | 13 651 | 100 |
| Interpretation | 13 651 | 100 |

a: categories defined in Table 4
For each variable recorded during the 1999 serological survey on PPR in Ethiopia and stored in the database, the number of samples for which information was recorded and the % out of the total number of samples it represents are shown.

$$\rho = \frac{\sigma^2(b)}{\sigma^2(b)+\sigma^2(w)}$$

Where:

$$\text{Between-group variance} : \sigma^2(b) = \frac{\sum_{i=1}^{K} n_i(\bar{x}_i-\bar{x})^2}{(K-1)}$$

$$\text{Within-group variance} : \sigma^2(w) = \frac{\sum_{i=1}^{K}\sum_{j=1}^{n_i} (x_{ij}-\bar{x}_i)^2}{(n-K)}$$

For a *wereda* in which *K kebelle*s are sampled with $n_i$ samples obtained from *kebelle i*

Chloropleth maps were produced using ArcGIS version 9.1 (ESRI, Redlands, California) to show the distributions of i) seroprevalence by *wereda* and ii) intra-*kebelle* correlation coefficient.

The hypotheses that species, age group and sex significantly differed between positive and negative animals were first tested in a univariate analysis by means of 2-tailed chi-squared tests without adjustment for clustering of observations within *wereda*. In a second step, a logistic regression model was used to assess the association between the potential risk factors sex, age and species and the outcome variable PPR serological status. The three independent variables were forced into the model. *Wereda* was included as a random effect to account for clustering within *weredas*. In the multivariate analysis, the first two age categories (less than 1 year and between 1 and 2 years) were collapsed into a single category due to the low numbers of observations in the "< 1 year" group. Associations were deemed significant when $P \leq 0.05$ by Wald test. The reliability of the regression coefficient estimates was assessed by testing the sensitivity of the quadrature approximation.

To assess whether the intra-cluster correlation was associated with the magnitude of the serological response of the animals in the *wereda*, we calculated non-parametric correlations (Spearman) between ρ and the 50$^{th}$, 75$^{th}$ and 90$^{th}$ percentiles of the ELISA inhibition percentage and between ρ and the seroprevalence.

Statistical analyses were conducted using SPSS 15.0 for Windows® (SPSS Inc., Chicago, Illinois) and Stata 9.0 (Stata Corporation, College Station, TX).

## Results

### *Seroprevalence of PPR in Ethiopia*

One hundred and two individual observations were dropped because of missing serological results. The remaining 13 651 individual observations were used in the analysis. The variables included in the dataset and the number of observations for which each variable was available are presented in Table 2. The periods of sampling, submission and analysis, based on the samples for which dates were available, lasted from 26 March 1999 to 5 June 2002, 19 October 1999 to August 2002 and 6 February 2001 to 13 February 2003, respectively.

The distribution of samples across regions and the prevalence per region are presented in Table 3.

**Table 3: Prevalence of PPR in the seven surveyed regions**

| Regions | Number of samples collected in each region and % of the whole survey | Prevalence with 95% Confidence Intervals |
|---|---|---|
| Afar | 1653 (12.1%) | 15.3% (13.6–17.0) |
| Amhara | 5992 (43.9%) | 4.6% (4.0–5.1) |
| Benishangul Gumuz | 729 (5.3%) | 8.0% (6.0–9.9) |
| Oromia | 2290 (16.8%) | 1.7% (1.2–2.2) |
| SNNPR | 1622 (11.9%) | 1.8% (1.1–2.4) |
| Somali | 465 (3.4%) | 21.3% (17.6–25.0) |
| Tigray | 900 (6.6%) | 15.3% (13.6–15.9) |
| Total | 13651 (100%) | 6.4% (6.0–6.8) |

Number of samples collected and prevalence of PPR in each of the surveyed regions. In brackets: % of the whole survey they represent and 95% confidence intervals.

There were large variations between the different regions in the distribution of the samples. Most of the samples have been collected in the northern part of the country and particularly in the Amhara region (43.9% of total samples collected). In regions like Somali or Afar there are about the same numbers of samples collected per *wereda* whereas in Amhara differences across *wereda* are more pronounced perhaps as a result of accessibility constraints. Similarly, few samples were collected in 6 *weredas* of Tigray region that had never been surveyed before.

There were important differences in the prevalence across regions, with the Oromia region showing the lowest prevalence (1.7%, 95% CI: 1.2–2.9) and the Somali region the highest (21.3%, 95% CI: 17.6–38.8) (Table 3). The variations are even more important for prevalence at the *wereda* level as shown in Figure 1. *Wereda* level prevalence estimates ranged from 0% for Guba in Benishangul region or Ab Ala in Afar region to 52.5% for Dolo Odo in Somali region. *Wereda* with the higher prevalence levels seem to be mainly those in areas of low altitudes where pastoral management systems prevail over sedentary ones.

### *Risk factors for positive serological status against PPR*

Descriptive statistics for the variables under study and the results of univariate comparisons are presented in Table 4. The proportions of seropositive animals significantly differ between species, age groups and sex categories. In the univariate analysis, sheep were 4.4 more likely and goats 5 times more likely to be seropositive for PPR than shoats (category combining both sheep and goats). Females were 1.3 times more likely to be seropositive than males. Regarding the age of the animals, none of the 41 animals younger than 1 year were positive. The highest prevalence was observed among animals older than 3 years, 12.6% of which were seropositive.

Results of the logistic regression assessing the relationship between species, age and sex and serological status are presented in Table 5. The only factor significantly associated with the odds of positive serological status was the age of the animal. Increasing age was associated with an increasing odds of seropositive status, with animals over 3 years old having almost twice the odds of been positive than animals under 2 years old.

As expected, there was strong evidence of significant intra-*wereda* correlation (intra-cluster correlation $\rho = 0.36$; $P < 0.001$). Despite the large number of observations per *wereda* (average of 166 animals) and the large intra-*wereda* correlation there was no evidence of unreliability of the quadrature approximation when estimates obtained using different numbers of cutpoints were compared.

### *Intracluster correlation coefficient ($\rho$)*

The 43 *weredas* for which the intracluster correlation coefficient ($\rho$) was calculated included between 2 and 9 *kebelles* each (median = 5) and these *kebelles* included between 15 and 180 individual animals each (median = 40). The estimated intra-cluster correlation coefficients are presented in Figure 2. Median $\rho$ was 0.029. Although the values seemed very heterogeneous, two groups can be clearly distinguished: One including nearly 80% of the *weredas* (34/43) with very low values of $\rho$ ($\rho < 0.12$) and the other with 9.3% (4/43) of the *weredas* showing a strong intracluster correlation, $\rho > 0.37$.

Figure 3 shows the geographical distribution of the values for $\rho$ by *wereda*. The map shows higher values for the correlation coefficient in the north of the country. The intra-*kebelle* correlation coefficient was highly correlated with the inhibition percentages among the animals sampled in the *wereda*: Spearman rank correlation coefficients of 0.45 ($P = 0.003$) with the median inhibition percentage, 0.45 ($P = 0.002$) with the 75$^{th}$ percentile of the inhibition percentage and 0.61 ($P < 0.001$) with the 90$^{th}$ percentile of the inhibition percentage. The intra-*kebelle* correlation coefficient was also highly correlated with the sero-prevalence found for each *wereda*: Spearman rank correlation coefficient of 0.67 ($P < 0.001$).

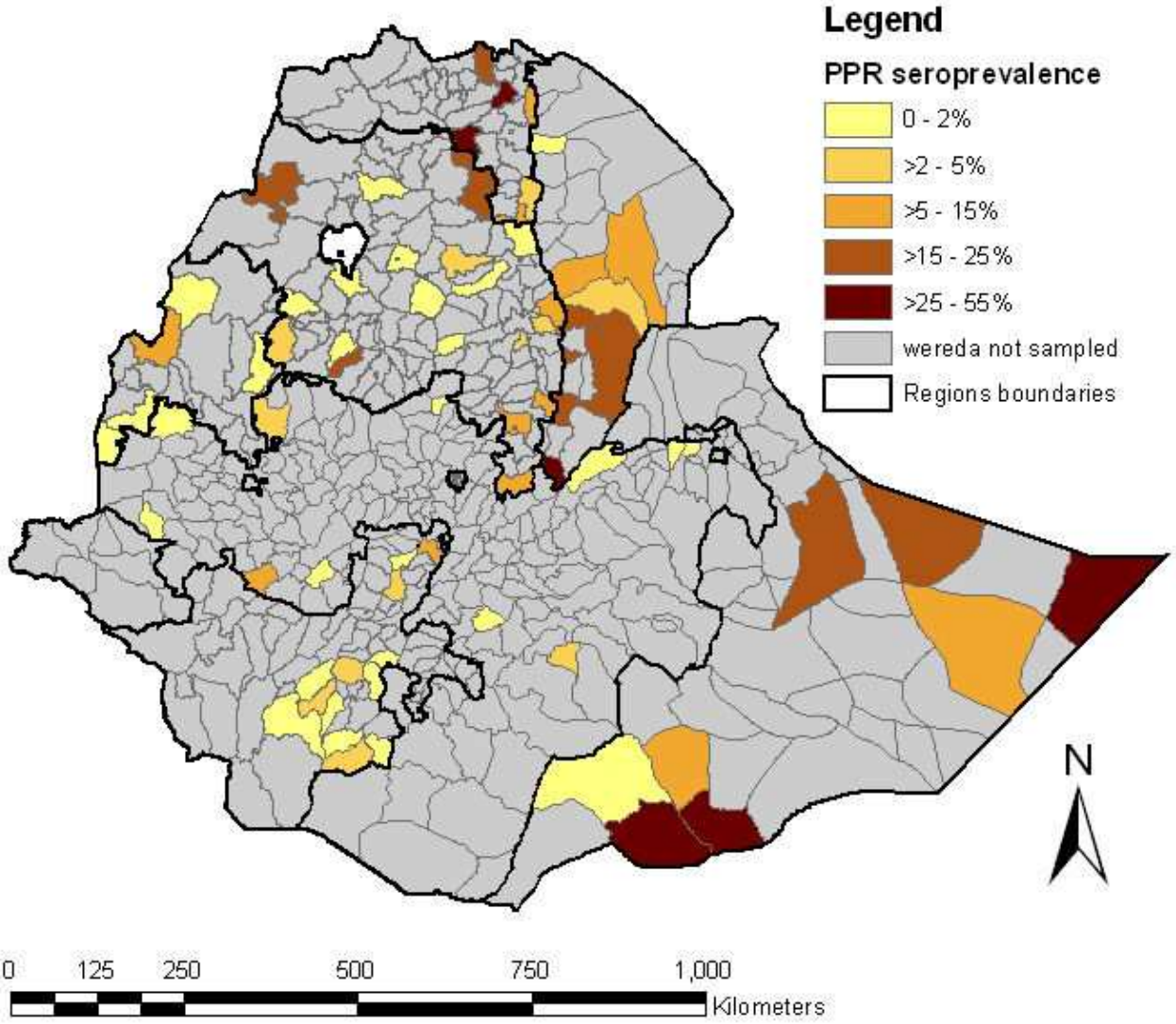


**Figure 1**
**Seroprevalence of PPR across wereda in Ethiopia**. Administrative map of Ethiopia indicating the regions and *weredas* boundaries. For each *wereda* seroprevalence of PPR was calculated by dividing the number of positive valid samples by the number of individual sampled in the wereda. As the colour gets browner higher is the seroprevalence found in the area. In grey, wereda for which no data was available.

## Discussion

Although estimates have to be interpreted with caution because it had not been possible to ensure that random selection was used at all sampling stages, the results indicate that PPR has been circulating in most of the country before large vaccination campaigns were implemented. Lack of large scale vaccination campaigns before the survey was conducted suggests that our seroprevalence estimates are likely to reflect infection [31]. Given the sensitivity and specificity of the test our results are likely to overestimate, slightly, the true proportion of seropositive animals [34,35]. On the other hand, by using the results of an imperfect test as indicators of true infection we are probably underestimating the true values of rho [36]. Despite an overall frequency of 6.4%, the seroprevalence of PPR was above 50% in some *weredas*. PPRV circulation before 2000 has been heterogeneous: areas of low altitudes appear to have suffered more from infection than areas of highlands. Reasons for this may be related to different production systems with exchanges and move-

**Table 4: Descriptive statistics of qualitative variables and univariate associations with seropositive status against PPR (two-tailed *P*-values for the $\chi^2$ – test of association).**

| Variable | Description | N | % of factor | % positive | P |
|---|---|---|---|---|---|
| **Species** | | 13 651 | 100 | | < 0.001 |
| | Sheep | 4 211 | 31 | 8.3 | |
| | Goats | 4 585 | 33.5 | 9.4 | |
| | Shoats* | 4 855 | 35.5 | 1.9 | |
| | Not available | 1 000 | 10 | | |
| **Age** | | 4 648 | 34 | | 0.003 |
| | Under 1 year old | 41 | 0.9 | 0 | |
| | Between 1 and 2 years old | 392 | 8.4 | 10.5 | |
| | Between 2 and 3 years old | 2 014 | 43.3 | 9.7 | |
| | Over 3 years old | 2 201 | 47.4 | 12.6 | |
| | Not available | 9 003 | 66 | | |
| **Sex** | | 5 868 | 43 | | 0.013 |
| | Males | 1 007 | 17.2 | 7.0 | |
| | Females | 4 861 | 82.8 | 9.4 | |
| | Not available | 7 783 | 57 | | |

* Sheep and goats not being distinguished

Results of 2-tailed chi-squared tests of the hypothesis that species, age group and sex differed between positive and negative animals. A description of each variable is presented including the number and % of each category in the sampled population and the % of positive. The proportions of seropositive animals significantly differ between species, age groups and sex categories (P-value (P) < 0.05).

ments in areas of lowlands being more frequent and involving larger numbers of animals. In Ethiopia small ruminants mainly thrive on free range pasture lands, shrubs and forest grounds. Agro climatic conditions influence the availability of these resources and the movement of animals becomes necessary in order to ensure the provision of fodder and water. This is particularly important during the dry season and in low altitude areas where resources are scarce. In addition, animals are exchanged between households and flocks as a result of social practices and changes in economic conditions that exhibit seasonal patterns. The seasonality of animal movements could partly explain the occurrence of the disease in Ethiopia mainly between the months of March and June [7,17,31].

**Table 5: Results of a logistic regression of sex, age and species on serological status against PPR with *wereda* as random effect.**

| Variable | OR | P | 95%CI |
|---|---|---|---|
| **Sex** | | | |
| male | ref | | |
| female | 1.15 | 0.37 | 0.85–1.54 |
| **Age** | | | |
| < 2 | ref | | |
| 2 – 3 | 1.27 | 0.23 | 0.86–1.88 |
| > 3 | 1.78 | < 0.01 | 1.20–2.62 |
| **Species** | | | |
| sheep | ref | | |
| goat | 1.08 | 0.50 | 0.86–1.35 |
| shoat | 1.42 | 0.51 | 0.50–4.04 |

Results of the logistic regression model used to assess the association between the potential risk factors sex, age and species and the outcome variable PPR serological status. *Wereda* is included as a random effect to account for clustering within *weredas*. The only factor significantly associated with the odds of positive serological status is the age of the animal (P < 0.01). Animals over 3 years old have almost twice the odds of been positive than animals under 2 years old.

Although the overall seroprevalence of PPR in Ethiopia appears to be low compared, for example, to the 22.4% reported in Turkey and 33% in India, it is difficult to draw any conclusions because of the differences in sampling procedures in the different studies that affect their representativeness [16,17]. However, a common feature described by the respective authors are heterogeneities, possibly related to agro-climatic and socioeconomic factors.

Age appears to be a risk factor for seropositive status, and its linear effect suggests that PPRV is highly immunogenic, naturally infected animals remaining positive for a long time.

The intra-*kebelle* correlation coefficient was found to be very low in most *wereda* with a small number of them showing high values. Differences in biological factors probably explain this variability. One hypothesis could be the past or recent circulation of PPRV reflected by a low or

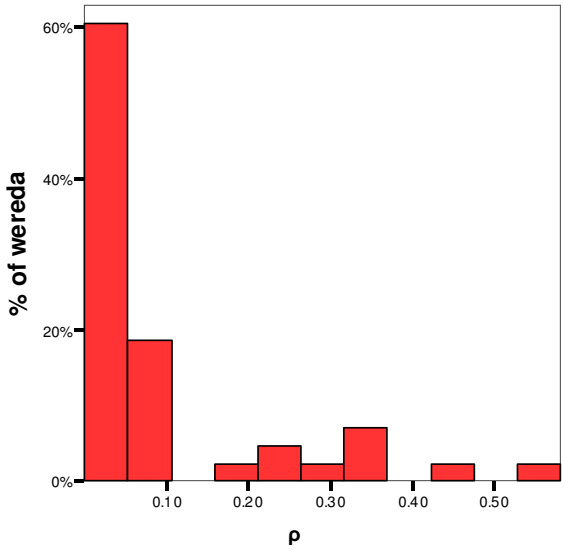


**Figure 2**
**Distribution of the correlation coefficient (ρ) across wereda**. Histogram showing the values of the intra-cluster correlation coefficient calculated for 43 *weredas* for which information about the *kebelle* of origin of the samples was available as indicated in the Methods section. Two groups can be distinguished: one including almost 80% of the *weredas* with low values of ρ (ρ < 0.12) and the other with 9.3% of the *weredas* showing a strong intracluster correlation (ρ > 0.37).

a high value of ρ, respectively, along with a low or high seroprevalence. Assuming that a high inhibition percentage could reflect recent infection, the strong correlation between ρ and inhibition percentage would be consistent with the interpretation of high ρ as being indicative of current or epidemic presence of the virus in a few *kebelles* within the *wereda*. This correlation then diminishes with time, diluting itself in a *wereda* as a consequence of a relatively rapid turnover of small ruminants (3 years), PPRV being highly immunogenic and that these are serological results. Considering that PPR is as a highly contagious disease and that both the within-and between-*kebelle* spread of infection determine ρ, the low value in certain *weredas* could also be attributed to *weredas* where animals of different *kebelles* mix a lot at market points or at water sources. The absence of an obvious spatial pattern in the distribution of ρ may reflect that spread of the disease has mainly occurred within individual *wereda* as opposed to large scale outbreaks involving several contiguous *wereda*.

Probabilistic sampling is a challenging task in a country with an infrastructure such as Ethiopia, since large areas have to be covered which are not easily accessible. Moreover, sampling frames of lower administrative units are often not available at central level. Under these circumstances, multi-stage sampling strategies such as the one used in the current study are the best option. Despite the random selection of *weredas* within regions, *kebeles* within *weredas* and villages within *kebeles* there is still potential for bias influencing our results due to non-random selection of regions and animals within villages. Although the large variation of values of ρ highlights the limitation of using a summary measure of ρ for a whole country as a basis for a sampling design, our estimates can inform the design effects needed to adjust for cluster sampling in future surveys on PPR in regions with similar production systems. As an example, if we consider the median ρ = 0,029, the standard sample size calculations using simple random sampling with 95% confidence interval for an estimated prevalence of 5% and an accepted error of 1% needs to be inflated by a factor of: D = 1 + 0,029(n-1), n being the average cluster size and D the design effect. That accommodates for the lack of independence between small ruminants belonging to a given *kebelle* [37].

Thus, to design a seroprevalence study at *wereda* level in Ethiopia, the clustering effect of the *kebelles* implies the sample size has to be increased by a factor of 1.26 if 10 small ruminants are to be sampled per *kebelle*, 1.55 if 20 small ruminants are to be sampled, 2.42 if 50 are sampled and 3.87 if a hundred small ruminants are to be sampled in each selected *kebelle*. Our findings are in agreement with other published values for ρ and D related to viral diseases or vector-borne infections. Thus the majority of ρ reported lay below 0.20 with widely varying estimates for highly contagious viral infections as Infectious bovine rhinotracheitis (IBR) [38]. To our knowledge no specific reference was available until now for peste des petits ruminants.

## Conclusion

Our study shows that PPR has extensively circulated across Ethiopia, but that there is large variation between regions and *weredas*. Although in most *weredas* there is a small variation between *kebelles* in some of them there are large differences that indicate the virus has only been introduced recently among some *kebelles* of the *wereda*, if our interpretation of high intracluster correlation coefficients as indicative of more recent introduction is valid, PPRV has been more recently circulating in the North of the country. The results of our study indicate that further research is needed to investigate the association of the presence of disease with the management practices in place. These findings are also important to direct future studies in other countries where PPR is of importance.

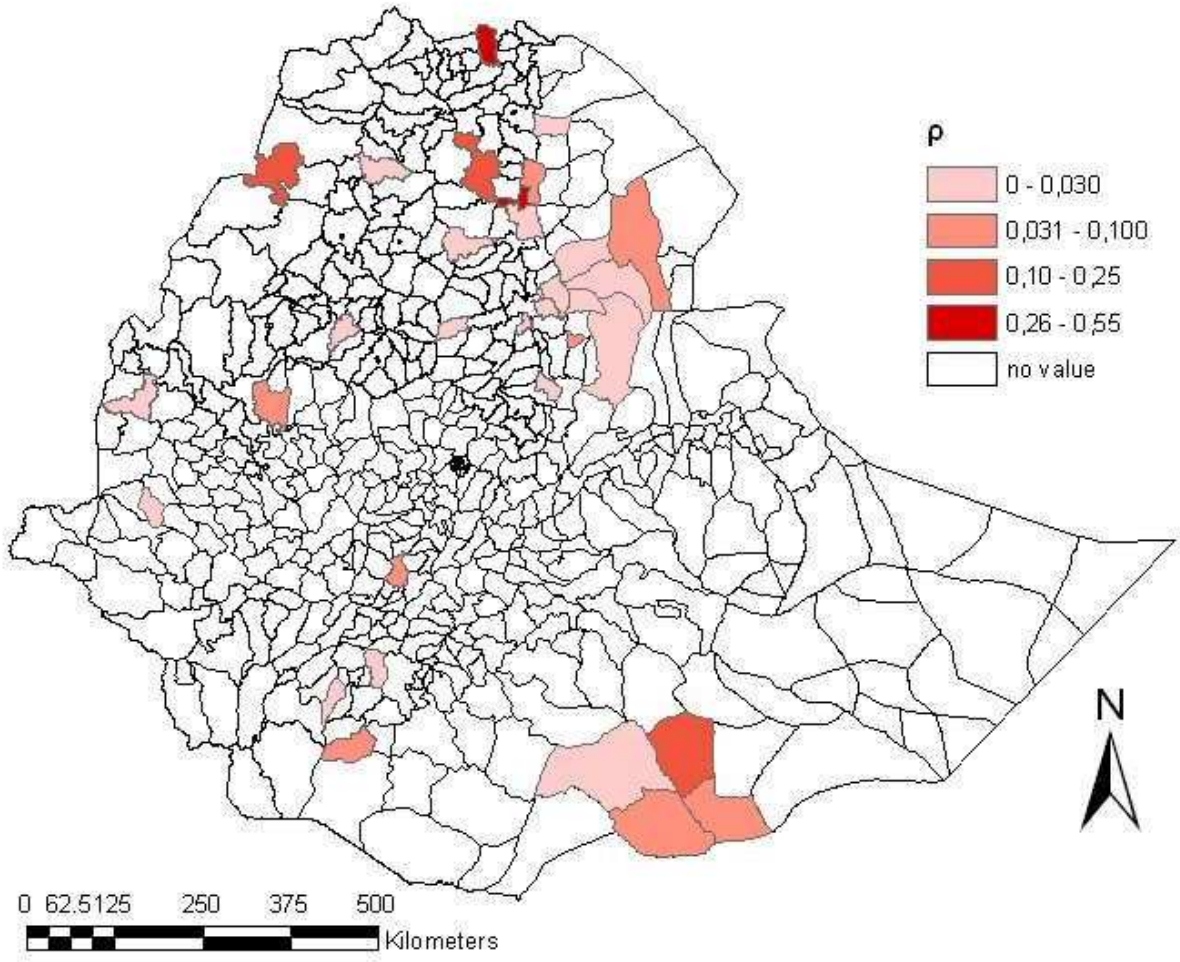


**Figure 3**
**Distribution of wereda ρ values across Ethiopia in 1999**. Geographical distribution of the values for the intra-cluster correlation coefficient (ρ) by *wereda*. The red is more intense in the *weredas* with a higher value for ρ.

## Abbreviations

ADB: African Development Bank; cDNA: complementary DeoxyriboNucleic Acid; cELISA: competitive Enzyme-Linked Immunosorbent Assay; EDI: ELISA Data Information; FAO: Food and Agriculture Organization; GDP: Gross Domestic Product; IAEA: International Atomic Energy Agency; IBR: Infectious Bovine Rhinotracheitis; NAHRC: National Animal Health Research Center; NLDP: National Livestock Development Project; OD: Optical Density; PI: Percentage Inhibition; PPR: Peste des Petits Ruminants; PPRV: Peste des Petits Ruminants virus; ρ: intracluster correlation coefficient.

## Authors' contributions

AWS, DC and LMY collected the data, created the electronic database and cleaned and processed the data for analysis. AWS and JG conceived and performed the data analysis. AWS drafted the manuscript assisted by JG. FR, DUP and GL raised funding for the study and assisted its coordination. All authors helped with the interpretation of the results and read and approved the final manuscript.

## Acknowledgements

We are grateful for the access to data and practical support given by the National Animal Research Health Center (Ethiopia), in particular its Director Dr Mesfin and Dr Abraham Gopilo. The authors would also like to thank Dr Berhe Gebreeziabher who facilitated the data collection and interpretation of results.

This study was carried out in the frame of the EU funded INCO-CT-2004-003670 RP/PPR MARKVAC project.

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*Epidemiol. Infect.* (2011), **139**, 875–885. © Cambridge University Press 2010
doi:10.1017/S0950268810001718

# Herd contact structure based on shared use of water points and grazing points in the Highlands of Ethiopia

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(*Accepted 24 June 2010; first published online 20 July 2010*)

## SUMMARY

The use of shared common water points (WPs) and grazing points (GPs) at two different levels of administrative aggregation (village and *kebelle*) in a region of the Highlands of Ethiopia was explored by means of a questionnaire survey and social network analysis. Despite GPs being more abundant than WPs (208 and 154, respectively), individual GPs provide more contact opportunities for animals. There was great variability in the contact structure of the selected villages within *kebelles* for both networks, with this variability being higher in the GP networks for each *kebelle*. Contrary to the commonly held view that WPs are critical for the potential transmission of infectious diseases, intervention at GPs in the Ethiopian Highlands may have greater impact on contacts and thereby opportunities for transmission of infectious diseases between flocks. Some villages appear naturally at much lower risk of introducing disease. These findings could help the design of surveillance and control activities for directly transmitted infectious diseases.

**Key words**: Contact, disease transmission, Ethiopia, livestock, social network analysis.

## INTRODUCTION

Ethiopia has the largest livestock population in Africa and it is ranked ninth in the world [1]. The very diverse geography/geology of the country defines several agro-ecological zones. The central part is characterized by a zone of highlands surrounded by a temperate transition zone that plunges into the central Rift Valley towards the south west. To the east, the lowland areas, i.e. zones of pastoral nomadic livestock husbandry are found [2].

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The climate is characterized by a long rainy season called *meher* from June to September representing about 75% of the annual rainfall, and a short rainy season called *belg* from February/March to April/May. The dry season extends from October to January [3]. Heavy rainfall during *meher*, low temperatures at the beginning of the long dry season and lack of water at the end of this season can be important constraints for agriculture and livestock production [4, 5].

Around half of the small ruminants in Ethiopia are found in the Highlands with a population mainly comprised of sheep. Production systems of goats are not well documented in that region but usually follow the same pattern as those of sheep where they occur.

Goats are mainly raised by traditional farmers together with other livestock in combination with agricultural activity. The mixed livestock–agriculture system with small herds present in the Highlands contrasts with the pastoral system found in the lowlands where larger flocks/herds are found [6, 7].

Grazing tends to be communal during the dry season and individual during the rest of the year with owners grazing the animals on their own land. A study by Larbodière [3] estimated that 90% of farmers mixed their animals with other flocks in the same village but most of them (80%) stop this practice during the long rainy season.

The process of 'villagization' during the mid-1980s in Ethiopia pushed farmers away from their plots of land. Moreover, in zones of intense cropping small ruminants shepherded mainly by children graze some distance away from the village [8].

Little is known about the contact structure of the farming population in developing countries, but it is likely to be complex and heterogeneous as a result of the need for continuous adaptation to variable environmental, socioeconomic, and institutional conditions. Management practices that favour contacts between animals from different origins in regions situated between the two parallels of 40° latitude north and south, are often used to explain the persistence of a number of directly transmitted diseases [9]. Mixing at watering points (WPs) or grazing points (GPs) has been identified as a key factor for transmission of diseases such as rinderpest, peste des petits ruminants (PPR) or foot-and-mouth disease (FMD) [10].

Social network analysis allows a description of the topology of the contact structure of livestock populations. The impact of network structures on the potential routes of transmission of infectious diseases can be investigated, provided that the network links are associated with known risk factors for disease transmission. Previous studies have shown the impact of such structures on the efficacy of surveillance and disease control programmes [11–13] using animal movements in intensive farming systems. However, the characterization of networks of animal contacts in settings without registered animal movements and structured animal contacts remains a challenge in terms of field network data collection and knowledge about the husbandry system. The aim of this study was to better understand the contact structure of the small-ruminant population in a selected area of Ethiopia by developing networks that reflect the natural heterogeneous mixing of flocks in a traditional mixed livestock–agriculture sedentary production system. The specific objectives were to describe, analyse and compare the contact networks generated through shared use of small ruminants' WPs and GPs in a region (*wereda*) of the Highlands in Ethiopia at different administrative levels (*kebelle*, villages) and to discuss the implications of such structures for the design of disease surveillance and control activities.

## METHODS

### Study site and sampling method

The study was conducted in the Bassona Werna *wereda* which covers 1020·35 km² in the central part of Ethiopia (Fig. 1). This area was selected because of its proximity to Addis Ababa (130 km northeast), and the availability of baseline production information from previous field studies [3, 14]. The study area represents the two agro-ecological settings usually found in the Highlands: *dega* from 2300 metres above sea level (m.a.s.l.) to 3500 m.a.s.l. (52%), and *woina dega* from 1500 to 2300 m.a.s.l. (48%). A stratified multi-stage sampling strategy was used, with the number of *kebelles* or villages set to a fixed number based on time and resource constraints. Ten out of 29 *kebelles* of the Bassona Werna *wereda* were preselected for the study according to two accessibility factors: the number of walking days necessary to reach them and the physical ability of the interviewers to reach the *kebelles*. The two most remote *kebelles* were included in a pilot study in which study protocol and questionnaires were evaluated, with the remaining eight being involved in the main body of research. In each of the eight *kebelles* used in the main study, a number of villages were randomly selected as listed in Table 1: 10 villages in five *kebelles*, 11 in two *kebelles* and eight in one *kebelle*. In Gudoberet *kebelle*, two villages out of the 10 selected were not accessible because of insufficient human resources and were replaced by one in Debele *kebelle* and one in Bere Ager *kebelle*, the only two *kebelles* with 11 villages in the study.

In each village, 10 small-ruminant owners were selected, using a systematic approach, for individual interviews [15, 16]. Starting from the centre of the village, the interviewer selected every second owner on a straight imaginary line heading north, then moved to the west, south and finally east until

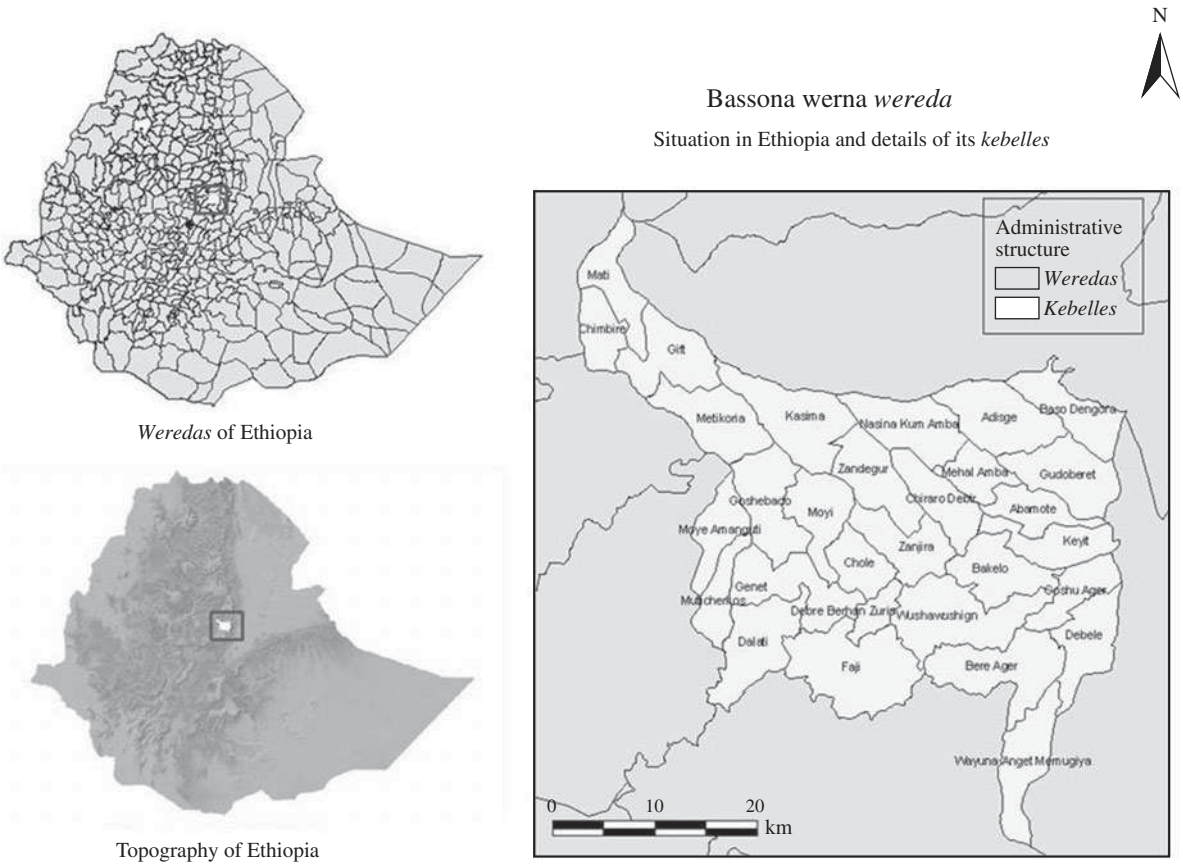


**Fig. 1.** Study site, location in Ethiopia and topography.

10 small-ruminant owners were identified. Given the average size of the villages this procedure ensured a representative selection of small-ruminant owners. In the evening of the day of the visit a separate questionnaire relating to WP and GP use was administered to a group of children in each of the 80 villages, because the children are usually responsible for taking the animals out for grazing after school and they generally spend time together when possible. In order to increase the reliability and validity of the answers, all available children from the village were assembled to complete one questionnaire per village.

### Field data collection

Two questionnaires (one for individual animal owners and another for the group of children) were designed and piloted, using the sampling method described above, in two *kebelles* selected for this purpose. Double-blind translation was used to validate the questionnaires in Amharic from their original English version. The owner's questionnaire was administered to individual farmers and included questions on flock/herd size and species composition and the possibility of the practice of *rebi* (keeping animals of other owners in return, e.g. for newborn lambs/kids). The questionnaire for the children focused on the names and time of use of GPs and WPs (long rainy season, short rainy season, long dry season, short dry season).

The questionnaires were administered by three interviewers (two of them being trained by the first one) from February to March 2007. During each visit, global positioning system location of the village was recorded along with the total number of households and the number of households keeping small ruminants.

### Data management and analysis

The results of the questionnaires were entered into Microsoft Excel® 2003 (Microsoft Corporation, USA). Descriptive statistics and tests for univariate associations between the variables representing altitude at which the household is located and species composition of the flock/herd were performed using SPSS for Windows version 15.0 (SPSS Inc., USA).

Table 1. *Density, isolates, mean degree of nodes, average geodesic distances and proportion of reachable pairs of nodes of common watering/grazing point networks of each selected* kebelle *from Bassona Werna* wereda *separately in February/March 2007, where node = villages and link = common watering/grazing point*

| *kebelle* (no. of villages) | Watering points | | | | | | Grazing points | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Density (%) | No. of isolates | Mean degree of nodes | Degree variance | Average geodesic distance | Proportion of reachable pairs of nodes (%) | Density (%) | No. of isolates | Mean degree of nodes | Degree variance | Average geodesic distance | Proportion of reachable pairs of nodes (%) |
| Abamote (10) | 11 | 3 | 1 | 0·6 | 1 | 11·1 | 8·9 | 4 | 0·8 | 0·8 | 1·4 | 15·5 |
| Angolela (10) | 33·3 | 0 | 3 | 2·4 | 1·4 | 53·3 | 11·1 | 4 | 1 | 1 | 1·3 | 15·5 |
| Bere Ager (11) | 7·3 | 7 | 0·8 | 1·1 | 1·3 | 10·9 | 14·5 | 4 | 1·5 | 2·4 | 1·8 | 38·2 |
| Birbisa (10) | 40 | 2 | 3·6 | 5·2 | 1·4 | 62·2 | 31·1 | 1 | 2·8 | 1·4 | 2·1 | 80 |
| Debele (11) | 5·5 | 6 | 0·6 | 0·4 | 1·2 | 7·2 | 14·5 | 5 | 1·5 | 2·6 | 1·5 | 27·3 |
| Goshebado (10) | 11 | 4 | 1 | 1 | 1·3 | 15·5 | 28·9 | 0 | 2 | 1 | 2·2 | 64·4 |
| Gudoberet (8) | 4 | 6 | 0·3 | 0·2 | 1 | 3·6 | 22·2 | 0 | 1·5 | 0·3 | 1·1 | 25 |
| Keyit (10) | 11 | 5 | 1 | 1·2 | 1·7 | 22·2 | 20 | 1 | 1·8 | 1·8 | 2 | 48·8 |

For $t$ tests, homogeneity of variance was assessed based on Levene's test for equality of variance and normality was assessed by examination of histograms. Statistical comparisons used $\alpha = 0{\cdot}05$ (two-sided).

Network data were entered as matrices using Microsoft Excel 2003. Several symmetric (undirected link) binary (presence/absence) networks were built with either *kebelles* or villages as nodes with the links being 'sharing WPs', 'sharing GPs'. Networks were also built with villages as nodes with 'sharing WPs', 'sharing GPs' as links for the dry and the rainy seasons. For each individual *kebelle* ($n = 8$) two symmetric binary networks were created with the nodes being villages and the links 'sharing WPs' and 'sharing GPs' within the *kebelle*.

For each network, density (proportion of all possible links that are actually present) and number of isolates (nodes not connected to any other) were calculated. For each node, degree was extracted, defined as the number of alternative sharing villages/*kebelles*. Average geodesic distances of the different networks defined as the mean number of links in the shortest path (geodesic) between all reachable pairs of nodes were extracted and used as an estimate of the overall power and speed of the network to transmit/diffuse. Whereas density, isolates, and degree primarily deal with adjacencies, the distance between nodes in a network helps to capture how cohesive and connected the network is.

Bootstrap paired-sample $t$ tests were applied to test for differences in density of two networks using 10 000 random permutations per test [17]. These tests are standard $t$ tests to compare the means of two groups (WPs *vs.* GPs, rainy *vs.* dry) but use a permutation test to generate the significance level so that standard assumptions of independence and random sampling are not required. Thus, they take into account that the 'observations' in network data are not 'independent' samples from a population. Through bootstrapping and permutations, null hypothesis distributions for the test statistics can be generated from the observed networks by using random assignment with thousands of trials [17].

The quadratic assignment procedure correlation function was used to calculate the correlation between two matrices, using the Jaccard coefficient based on 5000 permutations. Statistical tests of network data were conducted using UCINET 6.182 (www.analytic-tech.com/) and visualization of the networks with Pajek 1.21 (http://vlado.fmf.uni-lj.si/pub/networks/pajek/).

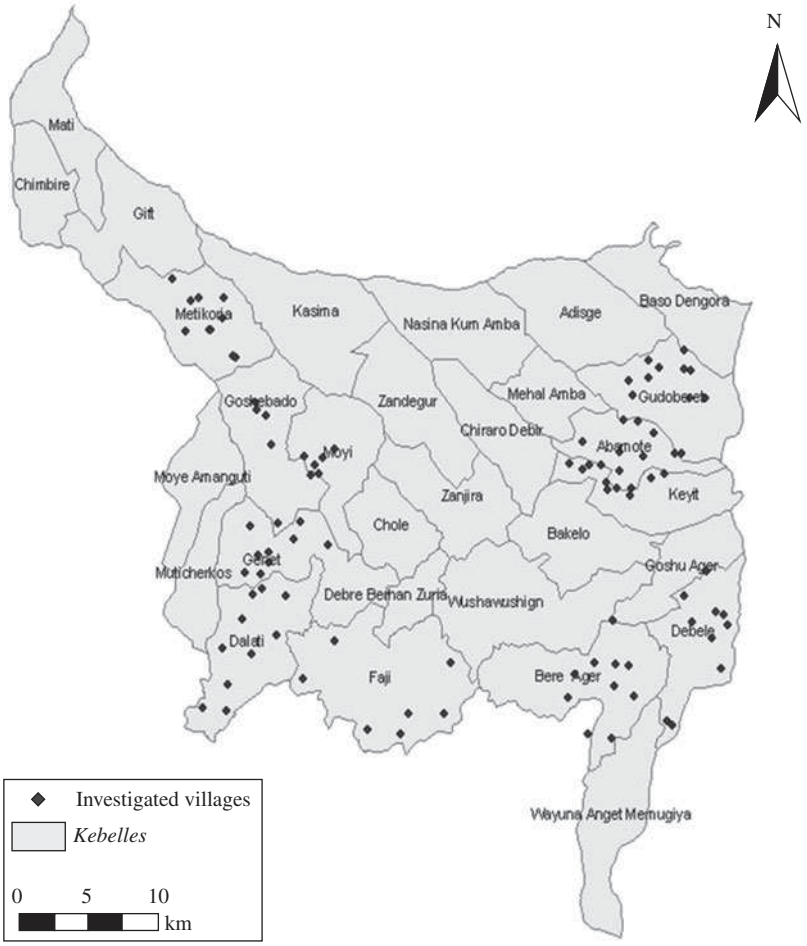


**Fig. 2.** Map of Bassona Werna *wereda* showing the administrative boundaries of all *kebelles* and the locations of the interviewed villages.

## RESULTS

### Questionnaire results

Data from 80 villages ranging in altitude from 2655 to 3336 m.a.s.l. were collected. On average 82% of the households investigated had small ruminants. Locations of the investigated *kebelles* and villages are shown in Figure 2.

The *kebelles* had a median of 1284 small ruminants [interquartile range (IQR) 1192–1313]. The villages had a median of 27 households with small ruminants (IQR 22–34) and the median flock had 10 sheep (IQR 6–15) and no goats (IQR 0–1). Sixty-nine villages (86%) had households rearing sheep and goats, nine (11%) had only sheep and the information was missing for two villages (2·5%). Villages keeping both species were situated at a significantly higher altitude than villages with only sheep (*t* test for independent samples: $P<0{\cdot}001$). In 27% of the interviewed villages at least one owner declared that they practised *rebi*.

Two subgroups of villages could be distinguished based on the geographical position in the *wereda* and altitude: one located west of the *wereda* including three *kebelles* (Goshebado, Angolela, Birbisa) with an altitude between 2500 and 2900 m.a.s.l. and the other east of the *wereda* including five *kebelles* (Abamote, Keyit, Bere Ager, Gudoberet, Debele) with an altitude between 3000 and 3300 m.a.s.l. During the interviews in the villages, 154 WPs and 208 GPs along with their period of use were identified.

### Network analysis

*At* kebelle *level*

The networks with *kebelle* as nodes and links based on 'sharing GPs' or 'sharing WPs' showed no isolates in their eight nodes and are displayed in Figures 3 and 4,

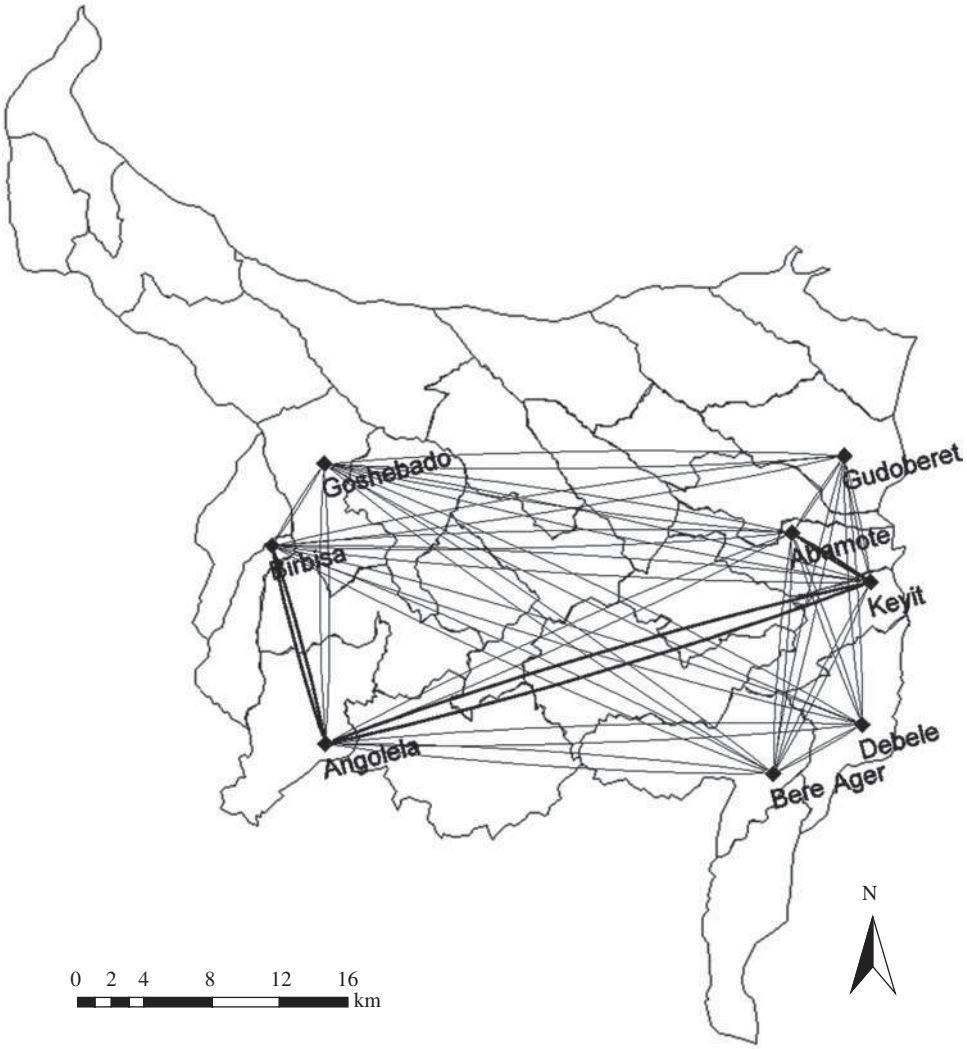


**Fig. 3.** Grazing points network with nodes being *kebelles*.

respectively. The average degree of the nodes in the GP network (6, range 5–7), was much higher than the same measure in the WP network (3·2, range 1–5) where there were two nodes linked to a single *kebelle*. Degree variance was much higher for the WP network (2·31) compared to the GP network (0·5) underlining a higher diversity of *kebelles* when comparing their number of links to other *kebelles* with WPs. The average geodesic distance of the 28 possible reachable pairs in a symmetric network of eight nodes without loops was higher for the WP network (1·7) than for the GP network (1·1) with 100 % of the pairs of nodes reachable in both networks. The density of the GP network (93 %) was also significantly higher than the density of the WP network (46 %) (bootstrap paired-sample $t$ test: $P<0{\cdot}001$). The Jaccard coefficient (0·467, $P=0{\cdot}013$) indicates that the shared use of GPs by *kebelles* is highly correlated with the shared use of WPs.

*At village level*

*Separate networks for each* kebelle. The WP and GP networks for each *kebelle* ($n=8$) with villages as nodes present a very variable structure between and within *kebelles*. Table 1 presents a summary of the extracted parameters for each of the 16 networks that were built. The isolates were more abundant in the WP networks compared to the GP networks.

The mean degree of the villages within *kebelles* was very variable in each of the networks, being more variable in the WP networks. The highest average degree of the nodes was found in Birbisa *kebelle* where villages were on average linked to 3·6 other villages of the same *kebelle* through WPs although with an

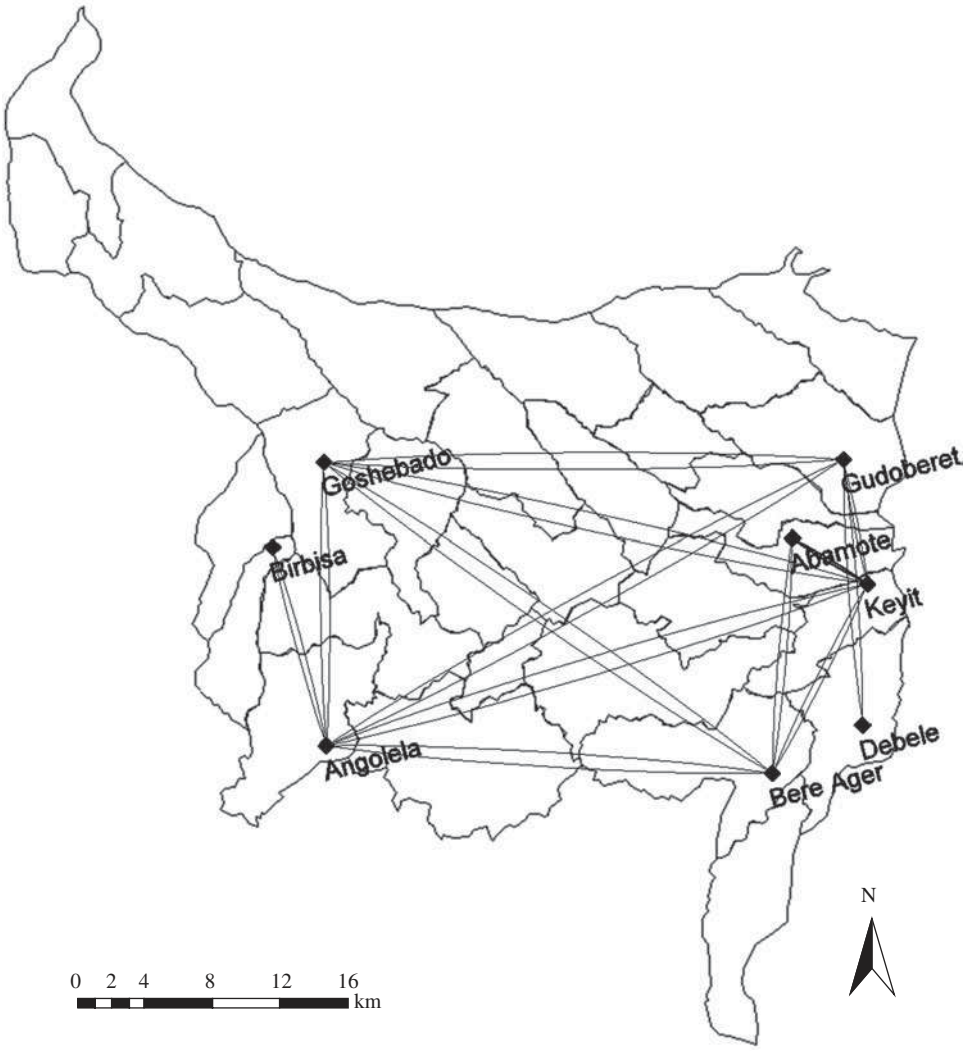


**Fig. 4.** Watering points network with nodes being *kebelles*.

important variability (degree variance 5·2). Birbisa was also the *kebelle* where villages had the largest average degree (2·8) in the GP network.

In general, density was higher in the GP networks compared to the WP networks with the highest values found in the Birbisa *kebelle* networks with 31·1% and 40% of all possible links present, respectively.

The average geodesic distance was higher for each of the GP networks except in the case of Angolela, which was the opposite. As the proportion of reachable pairs used in the calculus of this parameter was larger for all GP networks (except for Angolela) the cohesion of the GP networks in general are higher than the WP networks confirmed by the density and the number of isolates (Table 1).

*Network including all villages.* The WP and GP networks using villages had 80 nodes each. Table 2 shows the number of isolates, the density, the degree (range, average, variance), the average geodesic distance and the proportion of reachable pairs for the two networks considered.

Both networks showed low connectivity, with a density of 2·6% in the WP network and 5·7% in the GP network. The number of isolates when the links were based on shared WP use was almost double the number of isolates based on shared GP use.

On average villages were directly linked to two other villages by means of shared WP use (variance 4·2, range 0–11) and 4·6 other villages via shared GP use (variance 19·2, range 0–22).

The significant difference in density observed at *kebelle* level was confirmed at village level ($P<0{\cdot}001$) with the density of the GP network being higher than the density of the WP network. The Jaccard coefficient (0·259, $P<0{\cdot}001$) also confirmed that villages

Table 2. *Isolates, density, degree* (*including average and variance*), *average geodesic distance and proportion of reachable pairs of common grazing/watering point networks in the Bassona Werna* wereda *of Ethiopia in February/March 2007, where node = village* (*n = 80*) *interviewed and link = sharing watering/grazing point, taking into account seasonality*

| | Season | No. of isolates | Density (%) | Degree | Average degree | Degree variance | Average geodesic distance | Proportion of reachable pairs of nodes (%) |
|---|---|---|---|---|---|---|---|---|
| Watering points | All | 20 | 2·6% | 0–10 (Dalati) | 2·0 | 4·2 | 2·4 | 8·2 |
| | Rainy | 30 | 2·2 | 0–10 | 1·7 | 3·9 | 1·9 | 5·3 |
| | Dry | 24 | 2·4 | 0–10 | 1·9 | 4·1 | 2·2 | 7 |
| Grazing points | All | 11 | 5·7% | 0–21 (Dibut) | 4·6 | 19·2 | 2·9 | 65·9 |
| | Rainy | 19 | 4·5 | 0–19 | 3·6 | 14·3 | 3·3 | 50·5 |
| | Dry | 11 | 5·1 | 0–14 | 4 | 12·8 | 3·1 | 63·9 |

with contacts through shared GP use were more likely to also share WPs.

The average geodesic distance was higher in GP (2·9) compared to WP (2·4) networks. Yet again the proportion of reachable pairs is on average eight times higher in GP networks than in WP networks.

Each of the two networks with villages as nodes (considering 'sharing GPs' and 'sharing WPs') were then split into two networks in order to take into account the seasonality of the links between villages (rainy season and/or dry season). Table 2 reports the same extracted parameters for the networks of links during the rainy and dry seasons for the GP and WP networks.

In general rainy and dry GP networks have less isolates, higher average degree, larger degree variance, larger average geodesic distance, and larger proportion of reachable pairs than the corresponding WP networks. The density of the WP network during dry season was significantly higher than during rainy season (difference in density 0·003, bootstrap $t$ test two-tailed: $P = 0{\cdot}04$), which was not the case for the GP networks ($P = 0{\cdot}22$). The rainy and dry season networks were significantly correlated for WP and GP cases (Jaccard coefficient 0·906, $P < 0{\cdot}001$ and Jaccard coefficient 0·77, $P < 0{\cdot}001$, respectively), indicating that villages sharing WPs and GPs during the rainy season are more likely to also share them during the dry season.

## DISCUSSION

The aim of this study was to describe and compare the networks for small-ruminant flocks generated by shared use of WPs and GPs at different administrative levels in the Highlands of Ethiopia. To our knowledge it is the first time a field study of this kind has been conducted in Sub-Saharan Africa.

It should be noted that the purposive selection of *kebelles* within the *wereda* based on accessibility criteria may have resulted in some degree of bias limiting the ability to generalize the results. Thus our results should be interpreted as representative of the shared use of WPs and GPs in the studied *kebelles*. Since the proximity and adjacency of the *kebelle* and village boundaries have not been taken into account, some parameters describing the networks and the nodes may have been affected, but they still allow comparisons across the constructed networks. Although the questionnaires were administered once at the beginning of the rainy season, patterns of seasonal variation could also be addressed by using retrospective information. Given the impossibility of validating responses, the potential for recall bias, a weakness common to any questionnaire-based survey, was minimized by conducting group interviews of children and allowing the participants to reach an agreement upon the answers given [9]. Results obtained regarding small-ruminant population structure and management practices were similar to those reported 15 years ago [3, 6]. The relatively homogenous structure of the small-ruminant farms across the *kebelles* and villages interviewed suggest that the GPs and WPs frequented would not be dependent on the herd size parameter in our study.

Despite GPs being more abundant than WPs, individual GPs appeared to provide more contact opportunities for animals from different *kebelles* and villages as shown by the lower number of isolates,

higher density, and degree values of the corresponding networks. Expansion of cultivated land forcing farmers to increase mobility when searching for GPs shared by multiple villages could explain this result. This effect was stronger in villages located in the highest altitude areas of the Highlands which tend to be more isolated and rear mixed flocks of sheep and goats. As expected the WP network was denser during the dry season when some of the WPs become unavailable with less isolates, with temporary WPs close to the villages probably disappearing. The increase of average degree could then possibly be explained by villages redirecting their flocks to common WPs and establishing links with other villages.

There was great variability in the contact structure of the networks of villages within *kebelles* for both networks, in terms of number of isolates, degree and degree variance. Some villages did not share any WPs or GPs with other neighbouring villages suggesting that these villages would be naturally at much lower risk of introducing disease with geographical boundaries such as mountains or waterways or both possibly preventing contacts. The size of the areas (WP or GP) could also partly explain the number of flocks/herds sharing them although the spatio-temporal boundaries of the GPs and WPs could not be fully addressed in this study given the logistical difficulties in locating and measuring them. The average geodesic distances were in general larger in GP networks, taking more steps to reach a village from any other one. However, given the higher compactness of these networks, the proportion of reachable pairs due to the presence of greater components determines that GPs present a potentially greater ability to transmit/diffuse compared to WPs.

The variability was also observed at *kebelle* level. Bere Ager, Birbisa, Debele and Abamote appeared to be highly connected via GPs but not so through WPs. Some showed very low connectivity between their villages but were more central when considering the links to other *kebelles*, e.g. Bere Ager. According to values of the geodesic distance and the proportion of reachable pairs, GPs could be considered again as riskier for disease transmission compared to WPs. In summary and in the light of these results, GPs appear to be more important than WPs as contact points for small ruminants in the Highlands of Ethiopia at village and aggregated *kebelle* scale. Both scales were explored with no previous knowledge as to whether either the *kebelle* or the village should be considered a more appropriate epidemiological compartment based on contacts. Contrary to the common assumption that congregation of livestock at WPs is critical for the potential transmission of infectious diseases, the results of the current study suggest that interventions associated with shared grazing areas in the Ethiopian Highlands may be more important for the contact between flocks. Moreover since small ruminants spend more time at pasture than drinking at WPs transmission of infectious diseases could be expected to be more facilitated at GPs, increasing the number of flocks/herds at the same place during the same period of time [18]. Rotation of the areas used for common and individual grazing in the Highlands and the relatively small size of the current study compared to the diversity and size of the country warrants cautious interpretation of the findings. Similarly the dynamic aspect of the links in the networks studied could not be totally captured by the questionnaire. Yet it is likely that improved awareness by farmers and veterinary services of the potential for disease transmission associated with shared use of grazing areas as well as promotion of biosecurity-conscious management of the grazing rotation may assist in the control and prevention of infectious diseases in small ruminants in the Highlands of Ethiopia [19]. Communal GPs of *kebelles* are therefore the most appropriate location for health interventions like vaccination campaigns. If marking of vaccinated animals is not well received by farmers, cards attesting the vaccination status of the flocks could be delivered as a compulsory requirement for future access to a particular common GP, for example. However, if limiting access to communal pasture might prove to be a difficult task, alternative use of these critical GPs could be proposed. For instance these points could be selected as sentinels for disease surveillance [20] or critical risk points where 'human and economic resources should be prioritized in order to confront biological disasters' [21]. It would be interesting to ascertain the disease status of some of the villages that appeared to be at a theoretically lower risk given their contact pattern as revealed by their position in the different networks. If the overall healthier status of these villages was confirmed, the outputs of this type of analyses could inform cost-effective risk-based surveillance and control activities at village level [22]. This particularly applies to diseases for which transmission is direct and the agent labile in the environment as PPR virus. When indirect transmission occurs with inanimate or live vectors and the agent is more stable, e.g. FMD virus or sheep

pox virus, these results should be reconsidered if other parameters have been included, for example management practices with the sharing of material at contact points or on the biology of the vector. Design of transmission models, useful as decision-making tools when looking at different possible strategies of control, could be improved by taking into account our network study if the transmission probability is low [23], as well as the frequency of contact, and that the random mixing hypothesis cannot be assumed [24–26].

There are other opportunities for contact between small-ruminant flocks, one being through markets [5, 9, 10, 27, 28]. Studies should be conducted to describe other contact networks and compare them with GPs and WPs with respect to their structure and likely impact on transmission of infectious diseases. Applying this method within the frame of a larger scale study could help improve the understanding of the contact opportunities and patterns, especially in transition zones between high-altitude areas where small-scale sedentary systems prevail, and lowland pastoral areas. Thus, further recommendations for the surveillance and control of diseases of economic importance for farmers such as PPR, sheep pox, goat pox, and FMD could be made. In conclusion and contrary to common belief, in the Highlands of Ethiopia GPs may offer more opportunities for flock mixing and contact than WPs. Some villages appear to have a much lower risk of introduction of disease as a result of not sharing WPS or GPs with others. Local patterns of contact through sharing of WPs or GPs should be considered in the design of surveillance and control programmes.

## ACKNOWLEDGEMENTS

The authors thank Desta Beyene for assistance in the field. This study was carried out as part of the EU-funded INCO-CT-2004-003670 RP/PPR MARKVAC project.

## DECLARATION OF INTEREST

None.

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# IV. DISCUSSION

## IV.1. Apport et complémentarité des modèles

En appui aux investigations épidémiologiques, la modélisation mathématique permet une meilleure compréhension de la propagation de l'agent pathogène et aide à la décision pour mieux définir des moyens de contrôle de la maladie.

Si le modèle de transmission SEIR présenté dans cette thèse comprend beaucoup de simplifications, des descriptions reconnues comme très simplifiées de systèmes se rapportant à des maladies infectieuses capturent souvent de manière efficace leurs principaux traits dynamiques (Grenfell et al., 2002) et fournissent des éléments pour la gestion (stratégies de vaccination) (Singer et al., 2011).

La fiabilité des modèles pour la prévision dépend de la qualité des données relatives aux schémas de contact et aux réseaux de transmission dans et entre les populations (Rohani and King, 2010).

Les enquêtes transversales sérologiques comme celle réalisée en Ethiopie en 1999 sont importantes en tant que pré-requis à une meilleure modélisation de la dynamique de menaces émergentes ou non (Miller et al., 2010 ; Rohani and King, 2010).

La mise en relation des résultats obtenus par l'utilisation de différents modèles affine également l'évaluation du risque : Les complémentarités entre les différents modèles présentés ici et leurs apports en terme de gestion du risque sont schématisés Figure 4.

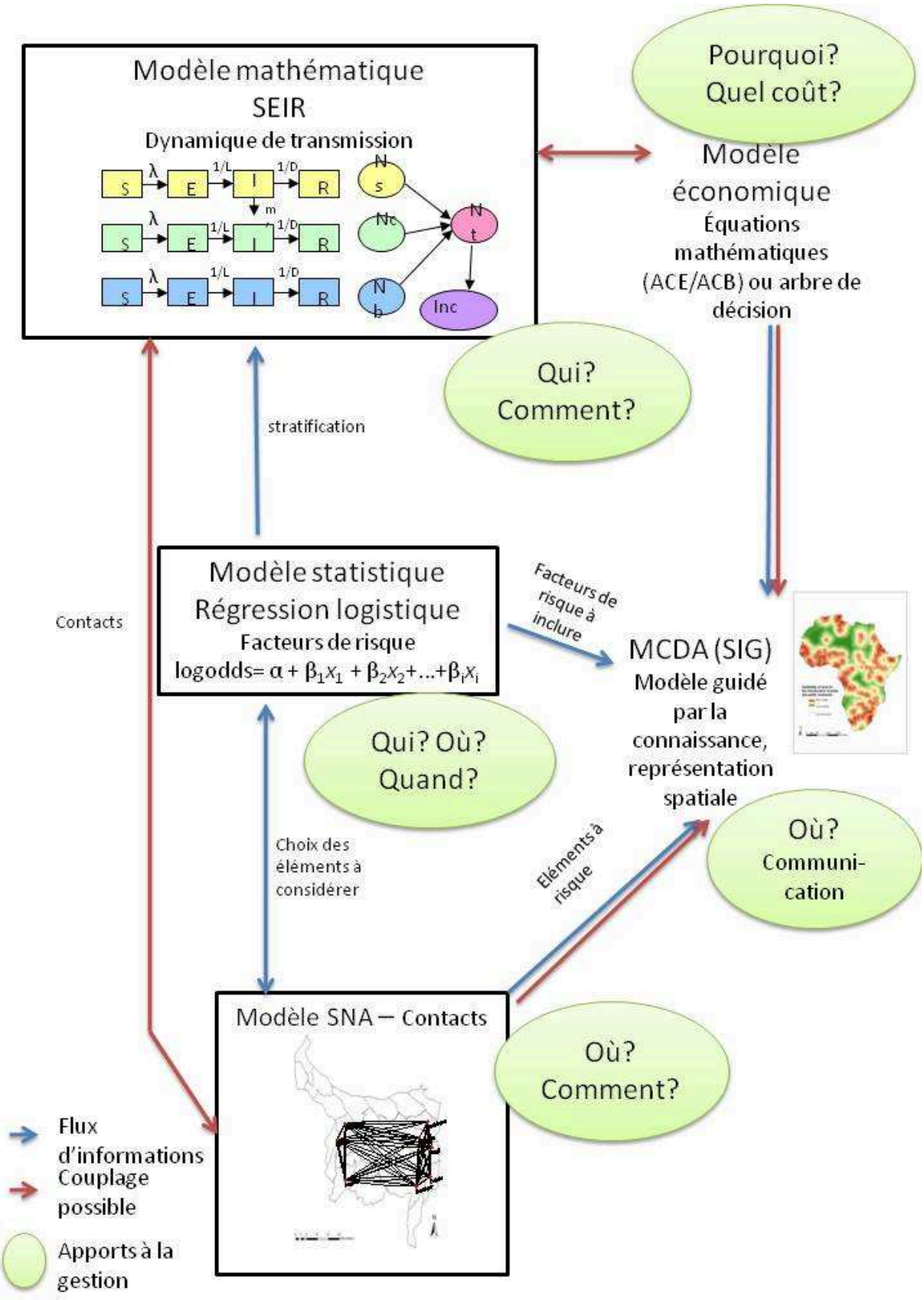


Figure 4: apports des modèles à la gestion du risque

Le modèle dynamique à compartiments (SEIR) peut permettre de caractériser le groupe d'individus auquel il faudrait s'intéresser mais aussi le niveau et le type d'intervention à mettre en place. En éclairant sur les conséquences telles la faisabilité ou non de l'éradication en fonction des programmes mis en place par exemple, ce modèle peut aussi permettre d'orienter les objectifs de surveillance ou de les revoir.

Le modèle statistique de régression logistique souligne lui aussi le ou les groupes d'individus à risque qui seront potentiellement à cibler mais il peut aussi permettre d'identifier d'autres facteurs de risque comme l'altitude ou la saisonnalité si les données relatives à ces variables ont été collectées et incluses dans le modèle. Dans notre cas les variables nous étaient imposées puisque l'enquête avait déjà été réalisée par les autorités ; mais a posteriori, on peut raisonnablement supposer que des données relatives à l'altitude auraient pu valider notre hypothèse, fondée sur le pattern d'occurrence de la PPR en Ethiopie, d'une maladie plus présente en régions pastorales de basse altitude que reprennent Megersa et al. (2011) qui concentrent leur étude sérologique dans ces zones.

Un autre intérêt de la régression logistique réside dans la hiérarchisation des facteurs de risque, un facteur de risque évident pouvant avoir une importance non significative ou moindre par rapport à ce que l'on aurait pu présumer au départ. Les résultats peuvent être intégrés par stratification du modèle à compartiments, permettre de poser les hypothèses pour la construction d'un modèle de réseaux sociaux avec le choix pertinent des nœuds et des liens à étudier, ou encore être représentés sous forme de couche d'information géographique pour un modèle d'analyse multicritère (MCDA) qui constitue un bon support de communication du risque compte tenu de la représentation visuelle qu'il permet. La modélisation des réseaux sociaux permet de renseigner la nature et les taux de contact. Ils sont donc intéressants à coupler avec les modèles de transmission SEIR dont une des principales limites réside dans la détermination des fréquences de contact qui varient au cours du temps (James, 2005). Ils permettent de répondre à des questions du type : Où privilégier les interventions ? Comment intervenir en fonction de l'importance de certains nœuds ou de certains liens dans le réseau ?

## IV.2. Développements complémentaires induits

Les résultats obtenus orientent vers la nécessité d'une meilleure compréhension de l'interface entre animaux domestiques et sauvages ainsi que de la sensibilité des camélidés (dromadaires en Afrique et chameaux en Asie) au virus et de leur rôle potentiel dans l'entretien et/ou la transmission de la maladie. Les filières et réseaux commerciaux mériteraient aussi d'être plus investigués. Des données similaires à celles de l'étude du partage des points d'eau et de pâturage mais se rapportant aux marchés sont en cours de valorisation (Annexe 1). Se pose aussi la question de l'échelle géographique. Une vision plus large qu'intra-pays ou même nationale semble indispensable dans un monde 'globalisé' (Awa and Achukwi, 2010 ; Arzt et al., 2010).

Si les hétérogénéités sont amplifiées lorsque l'échelle augmente, allant de pair avec le risque de simplifications erronées et avec une généralisation difficile, l'approche continentale voire par écorégions semble intéressante (Zinsstag et al., 2010). D'autre part, la distribution spatiale du risque de maladie et sa représentation visuelle sous forme de carte de risque peut aider à l'élaboration de stratégies de surveillance et de contrôle ciblées. Cette approche est particulièrement utile dans les situations où des données empiriques ne sont pas disponibles en tant que telles (Clements et al., 2006), ou quand les données ne sont disponibles que pour certains aspects d'une maladie multifactorielle au sens éco-épidémiologique (Tachiiri et al., 2006).

Ce sont ces facteurs qui ont orienté une autre étude en cours présentée ci-dessous employant une méthode spatialisée de modélisation d'analyse multicritères (MCDA) qui utilise les données sur des facteurs de risque connus pour déterminer les endroits où une maladie est la plus susceptible de survenir (Pfeiffer et al., 2008). C'est ainsi un exemple d'approche statique de modélisation guidée par la connaissance qui peut être utilisée pour produire des estimations qualitatives ou quantitatives de risque basées sur la compréhension des relations causales connues ou supposées conduisant à l'occurrence de la maladie (Pfeiffer et al., 2008).

La méthode combine les savoirs et les observations provenant de nombreuses sources différentes qui incluent : des articles publiés, des observations de terrain, l'expérience d'experts, et les représentations géographiques de facteurs de risque associés à l'introduction et à la dissémination de la maladie. Elle est guidée par notre meilleure compréhension épidémiologique des différents facteurs associés avec un risque accru d'avoir la maladie. Elle comprend huit étapes analytiques incluant la définition des facteurs de risque et la relation de chacun d'entre eux au risque, la recherche de leur représentation sous forme de cartes digitales qui seront standardisées afin d'être comparées et la combinaison des facteurs pour obtenir une estimation pondérée finale du risque pour chaque localisation du lieu d'étude (Pfeiffer et al., 2008).

Suivant cette approche nous avons cherché à identifier les endroits en Afrique à fort ou à faible probabilité d'introduction de la PPR et ceux à potentiels élevé ou faible de dissémination de la PPR une fois sur le continent.

Les cartes digitales correspondantes aux facteurs de risque cités dans la littérature (Tableau 1) furent recherchées sur le Web. Lorsque celles-ci n'étaient pas disponibles mais que les données le permettaient les cartes furent produites (c'est le cas des densités de moutons, chèvres, dromadaires et services vétérinaires) ; sinon le facteur était éliminé (dans le cas du facteur de risque 'race' par exemple) ou un facteur proche ayant une relation simple avec le facteur initial était utilisé, appelé 'proxy' (avec les zones sèches ou semi-sèches représentant les zones de nomadisme par exemple où il y a d'importants mouvements animaux et où les vols d'animaux sont plus fréquents, deux facteurs de risque identifiés par la recherche initiale dans la littérature) .

Afin de hiérarchiser les facteurs de risque entre eux on sollicita l'avis des experts. Ceux-ci furent sélectionnés sur la base de leur participation en tant qu'auteur à au moins deux publications en rapport avec la connaissance des facteurs de risque de la maladie. Les réponses de quatorze experts (sur les 22 identifiés initialement) classifièrent par ordre d'importance décroissante pour l'introduction de la maladie : la proximité aux routes puis celle aux ports et enfin aux aéroports, les trois facteurs représentant les voies d'entrée possible utilisées pour le commerce de petits ruminants domestiques ou d'ongulés sauvages. Pour la dissémination de la peste des petits ruminants une fois introduite on obtint dans

l'ordre : la densité de chèvres, de moutons, la distance aux services vétérinaires, la proximité de rivières navigables, la distance aux routes ayant la même importance que la densité de chameaux présente, la distance aux zones arides ou semi-arides, la distance aux villes, la distance aux aires protégées et enfin la proximité avec des voies ferroviaires.

Les cartes résultantes sont présentées ci-après, la carte de probabilité d'occurrence (Figure 7) résultant de la combinaison non pondérée de celle d'introduction (Figure 5) et de celle de dissémination (Figure 6):

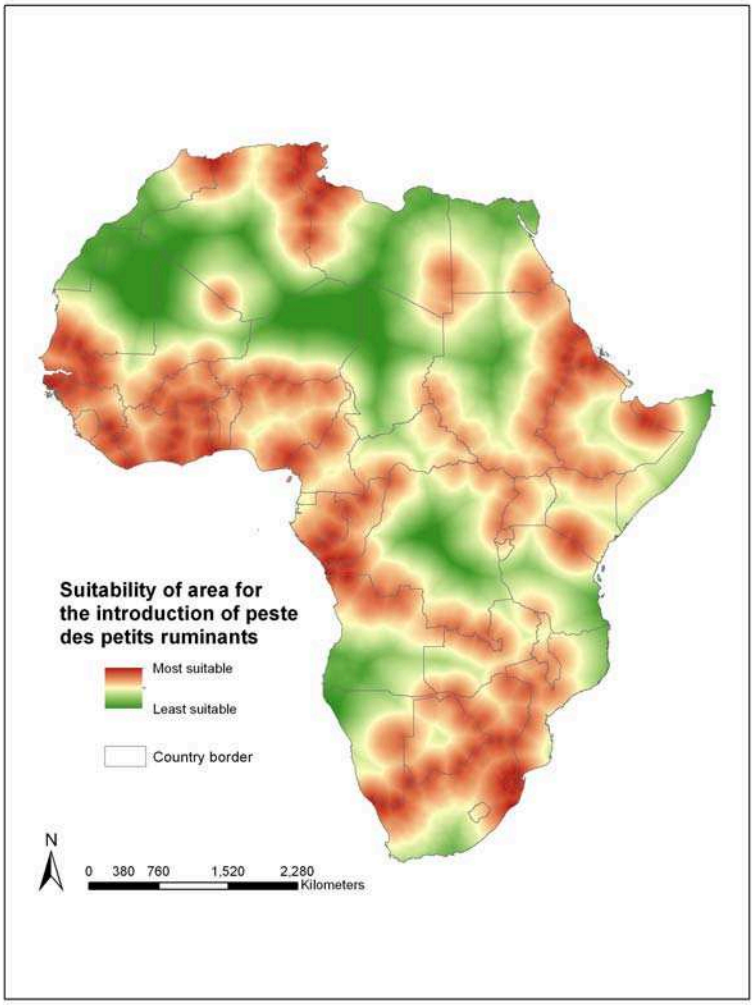


**Figure 5 : Localisations géographiques adaptées à l'introduction de la PPR en Afrique**

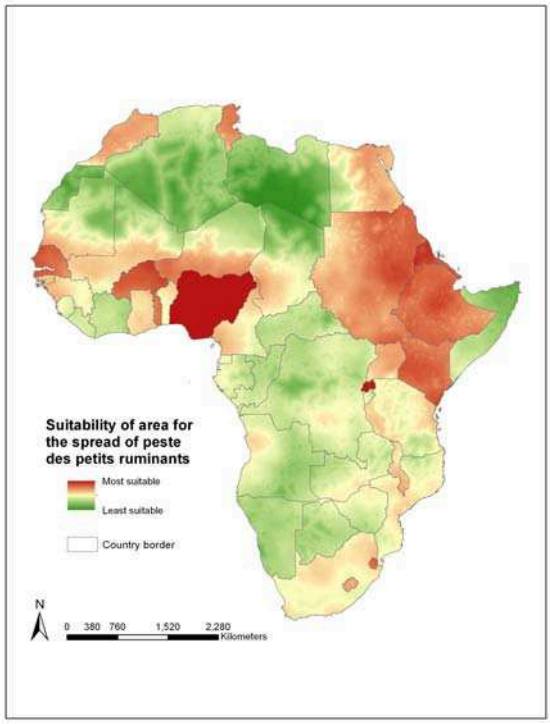


**Figure 6 : Localisations géographiques adaptées à la dissémination de la PPR en Afrique**

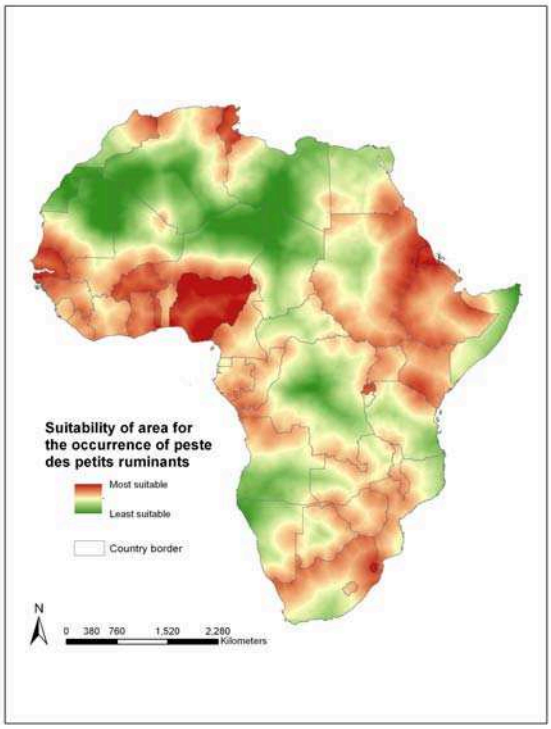


**Figure 7 : Localisations géographiques propices à l'occurrence de la PPR en Afrique**

On retrouve l'Ethiopie comme pays très favorable à l'introduction pratiquement tout au long de ses frontières et à la dissémination de la maladie particulièrement dans la zone des hauts plateaux. Par contre pour l'occurrence, la région des hauts plateaux semble s'éclaircir. Néanmoins il est difficile de conclure par manque de données pour valider le modèle aussi bien à l'échelle de l'Afrique que de l'Ethiopie (Annexe 2).

Pour l'interprétation des résultats d'une telle analyse, il est essentiel que l'utilisateur soit averti des hypothèses faites lors de la définition et de la quantification des intrants du modèle et de quelque biais potentiel en relation avec les sources d'information. Ceci est particulièrement important pour les modèles guidés par la connaissance. Ainsi on retrouve dans notre cas la délimitation géographique arbitraire des pays influencée par la présence des cartes de densité d'animaux et de services vétérinaires par exemple. Tous les facteurs de risque ne sont pas cartographiables ou disponibles sous une forme utilisable par le modèle. Le choix et l'utilisation de proxy peuvent être critiqués. De même pour les données à dires d'experts, à commencer par le choix des experts eux-mêmes. Cependant ces cartes doivent permettre d'engager la discussion avec les gens concernés et de souligner les besoins en données en étant spécifique sur le type de données à récolter.

## IV.3. Perspectives

L'appui scientifique est recherché pour répondre à des situations complexes pour lesquelles il existe un panel de réponses possibles. Ces circonstances impliquent une connaissance incomplète et le caractère prévisionnel des modèles est souvent limité. Cependant des décisions doivent être prises avec ou sans justification scientifique. Ainsi, le fait de pourvoir au contexte de décisions éclairées constitue déjà un objectif important de modélisation ; le résultat pouvant permettre de passer d'une prise de décision basée sur l'intuition à une prise de décision basée sur des faits (Singer et al., 2011).

Quelle que soit la sophistication de la méthode utilisée, la qualité des données d'entrée est un facteur limitant majeur du potentiel des modèles appliqués aux maladies infectieuses qu'ils soient spatialisés ou non, potentiel qui semble loin d'avoir atteint son maximum (Clements and Pfeiffer, 2009). Si beaucoup d'approches ont été développées pour utiliser au

mieux des données incomplètes ou sub-optimales, les épidémiologistes se doivent de travailler étroitement avec les responsables de santé publique à l'amélioration de la qualité des données de surveillance et dans l'idéal à la conception d'enquêtes de terrain ou de procédures adaptées à la méthode d'analyse appliquée pour leur interprétation (Clements and Pfeiffer, 2009). Les données utilisées sont en effet souvent inadéquates car elles n'ont généralement pas été collectées pour le type d'analyse effectuée par la suite et ont des limitations en termes d'utilisation condensée si les méthodes d'échantillonnage utilisées pour leur collecte sont différentes par exemple.

Outre la qualité des données, on doit également s'interroger sur le coût des programmes, leur évaluation et leur justification dans un contexte de ressources financières et humaines de plus en plus limitées notamment pour le recueil de données ciblées sur les groupes ou les endroits à risque (Stärk et al., 2006 ; Prattley et al., 2007). Si le travail présenté y répond en partie, une analyse économique de l'impact de la PPR à différentes échelles et utilisant différents points de vue mais aussi différentes méthodes de contrôle sont à envisager pour l'affiner (Rich and Perry, 2010).

L'évaluation économique concerne à la fois les coûts et les conséquences des activités mais elle éclaire aussi les choix des acteurs et compare plusieurs options : on peut la définir comme « l'analyse comparative d'options possibles, sur la base de leurs coûts comme de leurs conséquences » (Drummond et al., 1998 ).

Une approche économique par filières de plus-value ('value chains') relative aux petits ruminants pourrait éclairer sur les acteurs qui retireraient le plus de bénéfices de la mise en place de mesures de contrôle de la maladie et par la même seraient susceptibles de contribuer à leur financement (Bonnet, 2010 ; FAO, 2011).

Il est difficile de généraliser un type de surveillance à mettre en place ou à renforcer pour la PPR car les situations sont très diverses. Dans le contexte africain où la production de petits ruminants constitue un apport important à l'économie en terme de nombre d'animaux et de personnes impliquées dans les différentes filières, et compte tenu de l'impact (bien que non évalué précisément) qu'a potentiellement la PPR sur cette économie, la question semble importante. Dans le sud-est asiatique la situation est moins tranchée.

Par ailleurs, la problématique est différente s'il s'agit d'un pays atteint par la maladie ou encore indemne. Alors que les pays indemnes voudront sans doute se protéger de l'introduction de la maladie, les pays, comme l'Ethiopie, où elle est endémique, auront sans doute d'autres objectifs pouvant néanmoins être très variés et qui dépendront de sa place sur l'échelle de priorisation des problèmes de santé animale à résoudre. Dans tous les cas, il est essentiel que les objectifs de la mise en place ou de l'amélioration d'un système de surveillance existant tiennent compte des ressources humaines et financières disponibles.

On pourraient par exemple mener une étude coût-efficacité pour plusieurs options de niveaux différents de vaccination ce qui supposerait une évaluation préalable et de bonne qualité de l'efficacité du vaccin sur le terrain, même si celle-ci n'est pas remise en cause ici. En effet, si les niveaux sérologiques d'anticorps suite à l'administration du vaccin sont importants dans le suivi expérimental, l'analyse économique nécessite plutôt des résultats en termes de morbidité ou de mortalité évitées qui font aujourd'hui défaut. Cette évaluation pourrait compléter les résultats du modèle à compartiments qui interroge sur la pertinence de très hauts niveaux de vaccination. Si le seul moyen de contrôle est effectivement le vaccin, un objectif d'éradication ne semble pas réaliste et devrait donc permettre en retour d'éclairer les objectifs possibles de surveillance (Horzinec, 2011).

Le développement de tests sérologiques combinant plusieurs valences constitue une voie à explorer pour améliorer l'efficience des systèmes en place. Des recherches pour la mise au point d'un test permettant la détection de la Fièvre de la Vallée du Rift, de la fièvre catarrhale ovine, de la peste bovine et de la PPR sont en cours (Yeh et al., 2011). La mise au point de tests rapides pourrait en outre faciliter la détection précoce des foyers une fois dépassées les contraintes logistiques (Bruning-Richardson et al., 2011).

Cependant, considérant que la PPR peut être confondue avec d'autres maladies et que le virus de la PPR est considéré comme prédisposant certaines d'entre elles de type respiratoires, on pourrait envisager une surveillance syndromique telle que définie par Vourc'h et al. (2006) qui permettrait une économie d'échelle. Il conviendrait de regrouper les maladies cliniques en syndromes sur la base de signes/faits cliniques plutôt que sur celle d'un diagnostique spécifique. L'occurrence d'événements inhabituels nécessiterait alors des investigations complémentaires, comprenant la communication avec d'autres vétérinaires et

para-vétérinaires pour trouver d'autres cas, des études épidémiologiques ciblées, des projets de recherche ou des programmes de contrôle. On aurait alors un mélange d'approches de surveillance passive reposant sur une pyramide de remontées et de retours d'information dont la base est constituée par les éleveurs, et de surveillance active avec une population à échantillonner basée sur les facteurs de risque (Herholz et al., 2006 ; Thurmond, 2003). Si cette approche de surveillance syndromique était retenue, le développement de vaccins multivalents serait un atout complémentaire. Cependant les possibilités techniques et les contraintes locales conditionnent les valences à sélectionner ou à associer. Des approches de lutte avec un vaccin à double valence PPR-Fièvre de la Vallée du Rift sont développées en Mauritanie et en Tunisie (Ayari Fakhfakh et al., 2010 ; Faye et al., 2007).

Dans le cas de l'Ethiopie, où nous avons mené nos études sur le terrain et où la maladie est endémique, on peut s'interroger sur la pertinence d'une approche très coûteuse comme le séro-monitoring et en conséquence de la nécessité de développement d'un vaccin marqué permettant de distinguer les animaux infectés des vaccinés. En effet, des considérations économiques et le caractère endémique de la maladie orienteraient plutôt en première intention vers un système de surveillance passif où la sérologie pourrait éventuellement être envisagée avec parcimonie dans le suivi d'une couverture vaccinale. Les caractéristiques de transmission relativement aisée de la PPR avec une expression clinique sont un atout pour une approche dite passive et les considérations économiques laissent aussi peu d'alternative à la détection clinique des maladies des animaux d'élevage. D'autre part, les analyses de laboratoire ne sont pas faites fréquemment ce qui nous conduit à privilégier la surveillance syndromique. Quoiqu'il en soit des points de rassemblement tels les points de pâturage en particulier ou les points d'eau mais aussi les marchés ou les abattoirs apportent des opportunités pour la surveillance. L'identification de zones ou de production à risque peut permettre de savoir ou concentrer les efforts pour sensibiliser à la reconnaissance de la maladie ou à sa présence afin d'améliorer le système (Doherr and Audigé, 2001). Cette sensibilisation pourrait notamment être évaluée au démarrage ou à intervalles réguliers avec des approches participatives qui ont fait leur preuve notamment en fin d'éradication de la peste bovine dans les pays de la Corne de l'Afrique (Jost et al., 2007 ; Horzinec, 2011). Le niveau vaccinal à appliquer doit être aussi réfléchi en termes de répartition temporelle et

spatiale. Une vaccination en urgence telle que pratiquée actuellement autour des foyers déclarés lorsque les ressources sont disponibles, pourrait être potentiellement optimisée par la mise en place de 'barrières' vaccinales avec une administration plus régulière en lien avec la géographie du pays, à l'interface entre régions pastorales et plus sédentaires par exemple.

Par ailleurs, la prise en compte de l'écologie du virus PPR et des réservoirs de la maladie pourrait parfaire notre approche. L'écosystème impacte la dynamique des populations et la mobilité des hôtes qui influe sur la dynamique de la maladie. Un habitat plus fragmenté des hôtes pourrait expliquer un nombre et une intensité croissante de contacts à l'interface entre la faune sauvage et la faune domestique, potentiellement favorables à l'évolution du virus et au 'spillover' des souches.

Enfin, la biologie moléculaire et en première approche l'analyse spatiale de la variation génétique ('phylogéographie') qui a pour objectif de déterminer l'évolution génétique des maladies dans le temps et l'espace, permettrait de mieux décrire et d'analyser la distribution et l'émergence de la maladie (Real et al., 2005). Le séquençage et le typage des isolats de PPRV ont en effet apporté des perspectives intéressantes sur l'origine du virus mais aussi permis de suggérer par exemple que le virus était entré au Maroc en 2008 via le commerce d'animaux infectés provenant du Moyen-Orient et non par les mouvements nomadiques, ce qui avait été l'hypothèse première (Arzt et al., 2010). Cependant jusqu'à maintenant, les applications de phylogéographie pour la PPR (Banyard et al., 2010) restent descriptives ou exploratoires, limitées par la quantité d'isolats et de données disponibles et leur représentativité, ce qui fait que nous ne sommes pas dans un véritable contexte d'épidémiologie quantitative (Thiaucourt et Roger, 2005). Pour maximiser le potentiel de cette approche, l'analyse phylogéographique pourrait être couplée à des méthodes de statistiques spatiales ou d'approche de modélisation avec une composante spatiale explicite pour quantifier, expliquer et prédire la distribution de la PPR, mais cela reste un défi (Clements and Pfeiffer, 2009 ; Rohani and King, 2010).

# CONCLUSION

Les techniques de modélisation que nous avons présentées peuvent être utiles pour optimiser la surveillance en vue de renforcer la lutte contre la PPR. L'approche basée sur le risque est à privilégier dans un contexte où les ressources financières sont limitées en particulier dans les pays en voie de développement. La possibilité d'éradication de la maladie suggérée par ses similitudes avec la Peste Bovine dont l'éradication mondiale devrait être annoncée officiellement par l'OIE et la FAO cette année (2011) est à souligner. Cependant il y a un manque de données sur l'impact socio-économique de la maladie et le coût-efficacité des mesures de surveillance et de contrôle qui devra être comblé si les décideurs et les bailleurs de fonds doivent être convaincus de soutenir la lutte contre la PPR, aujourd'hui une maladie « négligée [2]» (Beyrer et al., 2007 ; AU-IBAR, 2010). Ceci est d'autant plus vrai que nous considérons que la menace reste avant tout limitée au 'Sud' – en effet, le 'Nord' a les moyens de maitriser rapidement cette maladie - et affecte principalement les éleveurs les plus pauvres, mettant pourtant en péril la sécurité alimentaire de millions de personnes. Si une implication mondiale parait utopique les approches régionales sont à encourager. Dans un contexte où malgré l'existence de moyens de diagnostic et de moyens de contrôle efficaces (vaccin avec protection pendant toute la vie économique de l'animal), la maladie continue son expansion, les approches en modélisation et d'évaluation, qui font défaut aujourd'hui sont à développer pour essayer de répondre à pourquoi/où/quand/qui/comment/à quel coût surveiller et contrôler/vacciner.

[2] D'après l'OMS les maladies négligées sont un groupe de maladies transmissibles qui sévissent dans les pays pauvres, participant ainsi à leur appauvrissement et dont l'impact bien que très important n'est pas forcément très visible et n'attire donc pas l'intérêt du public ou des médias.

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# ANNEXES

**Annexe 1 : Ortiz-Pelaez A., Guitian J., Yrjo-Koskinen A., Beyene D., Ashenafi G., Roger F., Waret-Szkuta A. Explaining the contact network of small ruminant farmers via livestock markets in the Ethiopian Highlands. To be submitted to the J R Soc Interface**

**Annexe 2 : Cartes du nombre de foyers de PPR déclarés à l'OIE par pays et par an en Afrique entre 1996 et 2011**

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JOURNAL OF THE ROYAL SOCIETY Interface

# Can geographical factors determine the choices of farmers in the Ethiopian Highlands to trade in livestock markets?



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|---|---|
| Journal: | *Journal of the Royal Society Interface* |
| Manuscript ID: | rsif-2011-0404 |
| Article Type: | Research |
| Date Submitted by the Author: | 22-Jun-2011 |
| Complete List of Authors: | Ortiz-Pelaez, Angel<br>Ashenafi, Getaneh; National Veterinary Institute (NVI<br>Roger, Francois; CIRAD, AGIRs Unit<br>Waret-Szkuta, Agnes; CIRAD, AGIRs Unit |
| Subject: | Computational biology < CROSS-DISCIPLINARY SCIENCES |
| Keywords: | network, market, ergm, Ethiopia, small ruminants, trade |
| | |

# Can geographical factors determine the choices of farmers in the Ethiopian Highlands to trade in livestock markets?

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## Abstract

Proximity and affiliation to the local market appear to be two of the most relevant factors to explain farmer's choices to select a particular trading point. Physical barriers may limit the options , especially in developing countries. A network of villages linked by traders/farmer-traders sharing livestock markets was built with field data collected in 75 villages from 8 *kebelles* in the Wassona Werna wereda of the Ethiopian Highlands.

Two exponential random graph models were fitted with various geographical and demographic attributes of the nodes (dyadic independent model) and three internal network structures (dyadic dependent model). Several diagnostic methods were applied to assess the goodness of fit of the models.

The odds of a edge where the distance to the main market Debre Behran and the difference in altitude between two connected villages are both large increases significantly so that villages far away from the main market and at different altitude are more likely to be linked in the network than randomly. The odds of forming an edge between two villages in Abamote or Gudoberet *kebelles* are approximately 75% lower than an edge between villages in any other *kebelles* ($p<0.05$). The conditional log-odds of two villages forming a tie that is not included in a triangle,

a 2-star or a 3-star is extremely low, increasing the odds significantly ($p<0.05$) each time a node is in a 2-star structure and decreasing it when a node is in a 3-star ($p<0.05$) or in a triangle formation ($p<0.05$)), conditional on the rest of the network.

Two major constraining factors, namely distance and altitude, are not deterrent for the potential contact of susceptible small ruminant populations in the Highlands of Ethiopia

**Key words:** network, market, ergm, Ethiopia, trade, small ruminants

## 1. Introduction

Livestock trade is an activity often occurring via an intermediate step in the form of a market or trading point with various levels of organization, procedures and control. An essential actor in this supply chain is the trader or middleman that represents a conduit between production sites and trading and consumption areas. For disease surveillance and control purposes, the interest lies in the different forms of interaction between production sites where susceptible animals are kept. This interaction usually occurs via a physical medium (fomites) and the actor/s (farmer, trader) for they can act as mechanical carrier of the virus, or move infected animals from an infected to susceptible farms. Markets have been shown to play a role in the dispersal of infectious diseases between livestock premises in countries with intensive and highly technified farming systems [1-2-3-4] although there is not much evidence of the drivers that take farmers to select a particular trading point.

Proximity and affiliation to the local market as part of the community network appear to be two of the most relevant factors to explain farmer's choices. Other factors like biosecurity, animal welfare and environmental compliance are not a priority for both farmers at the time to move livestock to markets and market operators as part of their business operations [5]. This risk-prone behaviour must be interpreted as the negative effect of the attempt to maximize the profitability of the farming enterprise. In developing countries the choices might be conditioned by market demands which usually operate initially at local level and the limited available resources in terms of transportation facilities. *A priori* it seems there is not much difference in the incentives that

farmers from different farming systems are presented with to discriminate between multiple trading points.

There has been a recent surge of research efforts to understand the pattern of animals movements and the role of livestock markets in developing countries, mainly due to their putative association with the spread and persistence of infectious diseases like H5N1 Highly Pathogenic Avian Influenza (HPAI) [6-7], FMD [8] and Trypanosomiasis [9], to name a few.

The network paradigm allows the integration of such interaction by joining actors represented by farms, production areas or animals and the trader or the trading point represented by the market. This phenomenon can be seen as a 2-mode or affiliation network [10] where the nodes are made of two distinctive classes: a set of actors (villages in our study) and a set of events (markets, traders); and the edges between nodes of different class that represent the choices of the farmers and/or traders to trade their small ruminants in a particular market or trading point. These are the basis of a bipartite graph that can be analyzed itself or transformed into other network structures.

Ethiopia has one of the largest population of small ruminants in Africa with 25 million of sheep and 23 million of goats in 2008 [11], distributed across a range of agro-ecological zones including a region of highlands in the central part of the country. Around half of the small ruminant population of Ethiopia are found in this area, mainly in small flocks [12-13]. In a recent study, the contact structure of small ruminant flocks in the Bassona Werna *wereda* (region) of the highlands of Ethiopia, based on the use of shared water and grazing points, has been described and analyzed [14]. Using the results of a survey carried out in the same area and within

the framework of the same project, the study presented in this paper aimed to investigate the trade patterns among small ruminant traders/farmer traders at village level with the view to set hypotheses on potential factors that explain the observed choices of markets, with special emphasis on the geographical barriers that traders/farmer-traders face at the time to trade livestock. By revealing the underlying structures of a contact network of production units represented by villages in the Bassona Werna *wereda*, these choices might be better understood. Alternative hypotheses about the observed contact structure and the underlying processes that generated it could be also postulated for further studies.

## 2. Materials and Methods

### 2.1 Sampling strategy and data collection

The highland town of Debre Berhan located in the Bassona Werna *wereda* at 2805 m above the sea level (m.a.s.l.) was used as a base for a set of research activities. The town is located 130 km North along the main road from the capital city of Addis Ababa. Ten out of the 29 *kebelles* of the Bassona Werna *wereda* were preselected for the study according to two accessibility factors: the number of walking days necessary to reach them and the physical ability of the interviewers to reach the *kebelles*. The two most remote *kebelles* were used to pilot the study protocol and questionnaires, with the remaining eight being involved in the main body of research. In each of the eight *kebelles*, 10 villages on average were selected and within each village, 10 small-ruminant owners were selected, using a systematic approach, for individual interviews. Details of the sampling strategy are described elsewhere [14]. During the visits, which took place in February-March 2007, global positioning system location of the village was recorded along with the total number of households and the number of households keeping small ruminants. For each

selected individual, the following demographic and trade behaviour data items were collected via a structured questionnaire: name of the trader/farmer-trader, name of the village and *kebelle* of origin, name and frequency of visits to the market for purchase and/or sale during the last year, average number of animals brought to the market, number of sheep and/or goats bought/sold last year, reasons for purchasing livestock, names of the *kebelles* crossed on their way to the market and whether they make a stop and mix with other flocks/herd in the *kebelles* they crossed. Additional attribute data of the villages identified by the interviewees were available from complementary studies in the area [14]. Questionnaire data were used to generate descriptive statistics for variables at village level assumed to reflect the flock contact structure mediated by livestock markets.

## 2.2 Analytical methods

### 2.2.1 Network definition

A symmetric binary 2-mode network was built linking villages and markets if trader / farmer-trader from a particular village reported to have operated in the market within the time window, i.e., during the year prior to the interview. The two-mode network is a bipartite graph that represents an affiliation network in which nodes of one class, the actors (villages), are linked to nodes of the second class, the events (markets) through the trading choices made by traders. This is so since traders / farmer-traders only traded animals from their villages of origin. The 2-mode network was converted to a 1-mode binary symmetric network of villages linked via trader/s operating in a common market during the time window.

### 2.2.2 The exponential random graph models (ergm)

A collection of *n* nodes linked via a set of relations (ties, links, edges) constitute a network. In network analysis not only the structure of the relational data can be of interest, but also the attribute characteristics of the nodes and of the edges can be important explanatory variables for the presence of the edge [15]. New developments in statistical network modelling allow researchers to move beyond the mere descriptive approach and test hypotheses about network structure [16]. One of them is a family of statistical models for generalised network inference, the exponential random graph models (ergm), developed as an extension of the first proposed log-linear model for network data: the $p^1$ model [17]. The exponential random graph models, referred to as p* models in the social network literature and developed during the 1990s by Wasserman and Pattison [18] as an extension of the Markov random graph [19], establish a general framework for the estimation of the probability that an edge is present in the network in the logit form, as a linear function of predictors, in a similar fashion as a logistic regression model. The particularity of these models is that the edge appears on both sides of the equation (as outcome and predictor) and often in multiple predictors, making the edge probabilities recursively dependent [20].

In the graph from which the network of this study is derived, the presence or absence of the edge between *n* number of villages (nodes) is defined by an adjacency matrix *Y* of dimension *n* x *n* so that

$$Y_{ij} = \begin{cases} 1 \text{ if the edge exists between node } i \text{ and node } j \\ 0 \text{ otherwise} \end{cases}$$

In general, the erg model specifies the probability of a random set *Y* of relations (edges and non-edges) given *y*, a particular set of relations among a set of nodes (villages), namely the observed

network, and their attributes, as a function of statistics that may depend on the network itself as well as covariates measured on the nodes , as described by [21]:

$$P_\theta(Y = y) = \left(\frac{1}{k}\right) exp\left(\sum_h \theta_h g_h\ (y,X)\right) \quad [1]$$

where

- $h$ is a configuration of the network represented by the observed set of edges among a subset of nodes of the graph containing them; different sets of configuration types represent different models (e.g. dyadic independence or dyadic dependent/Markov random graph) [25];
- $g_h(y, X)$ is a vector of statistics based on the observed adjacency matrix $y$, representing the structure of the network. $X$ allows for additional covariate information on the network. The model covariates could include raw network parameters like counts of the configurations in the observed graph (number of reciprocated edges, number of k-stars, number of triangles) but also node or edge-wise covariates like the distance of the village to a certain market or whether the edge is established between villages of the same *kebelle*, respectively. Each covariate should be a function of the observed data.
- $\theta_h$ are non-zero coefficients that denote the statistical parameter governing the probabilistic formation of the network. These are unknown parameters to be estimated.
- $k$ is a normalization constant and represents the quantity from the numerator summed over all possible networks, so that all probabilities sum to 1.

Eq. [1] can be re-expressed as the conditional log-odds (logit) of individual edges:

$$logit[P(Y_{ij} = 1 \mid n \text{ villages}, Y_{ij}^c] = \sum_{h=1}^{h} \theta_h \delta g_h(y,X) \quad [2]$$

where

- $Y^c_{ij}$ denotes all edges between nodes $i$ and $j$ other than the observed $Y_{ij}$ (the compliment of $Y_{ij}$ in $Y$), and
- $\delta g_h(y, X)$ is the amount by which $g_h(y, X)$ changes the log odds of an edge when the edge variable $Y_{ij}$ is changed from 0 to 1 (absence or presence of the edge).

The presence of $Y^c_{ij}$ in the conditional probability reflects the mutual dependence of ties. The logit formulation clarifies the interpretation of the $\theta$ vector: if forming an edge increases $g_h$ by 1, then the log-odds of that edge forming increase by $\theta_h$, with a single edge affecting in some cases multiple $g$ statistics [20]. A positive estimate means that the effect is more frequent in the observed network than expected by chance and a negative estimate means that the effect appears less in the network than it could be expected.

#### 2.2.2.1 Dyadic independent erg model

Network inference can be drawn assuming a dyadic independency whereby the state of the dyad (two nodes and their edge) depends on the attributes of the two nodes, for example, but not on the state of other dyads. Under this independency and when fitting these models, the vector of statistics $g_h(y, X)$ may always be calculated for $Y_{ij}$, regardless of the values of $i$ and $j$, without knowing anything about $Y$, in the case of an undirected network [22]. Given the difficulty for most networks to calculate the normalizing constant $k$, maximum pseudolikelihood estimation methods (MPLE) have been traditionally applied to estimate the model parameters assuming this conditional independence of the edge (for a review, see Wasserman and Robins [15]), superseded in the last few years by Markov chain Monte Carlo Maximum Likelihood Estimation

(MCMCMLE) techniques [23]. Models with only dyadic independent terms have a likelihood function that can be maximized using standard logistic regression methods, as shown above [24].

An initial dyadic independent exponential random graph model was fitted with the edge count as the only non-zero effect in the model, which corresponds with a Bernoulli random graph distribution, often called the simple random graph or Erdös–Renyi graph distribution [25]. To determine the variables in the final model, we used an iterative exploratory technique of progressively decreasing the model complexity by removing variables by decreasing order of p-values from the model containing the edge count and all other pre-selected covariates: pairwise difference in altitude of villages linked in the observed network, euclidean distance to the main market Debre Berhan (measured in decimal degrees), number of small ruminant farmers in the village, number of village sheep sold at the market, number of traders identified during the survey, *kebelle* (first order effect) and *kebelle* (second order effect). The model with the best fit (highest log likelihood) and more parsimonious was selected for reporting and diagnostics. Coefficients and p-values for each covariate and log likelihood and the Akaike Information Criterion (AIC) for the final model were extracted and displayed in Table 1.

**2.2.2.2 Dyadic dependent erg model**

When the likelihood of a dyad depends on the presence or the state of other dyads, the models to account for this dependency require computationally intensive estimation and imply complex forms of feedback and global dependence that "confound both intuition and estimation" [24]. The fitting of these models are based on an algorithm that draws on Markov Chain Monte Carlo

simulations (MCMC), a stochastic process that produce different results every time they are run, unlike dyadic independent models.

In order to describe the internal structure of the study network a dyadic dependent exponential random graph model was fitted using some of the traditional configurations in undirected networks that describe the structural cohesiveness of the network: the k-star (2-star and 3-star) and the triangle, apart from the edge count, as in the previous model. A 2-star is a subset of three nodes in which one node is connected to each of the other two, and a triangle is a subset of three mutually connected nodes. These configurations are defined hierarchically, so that a triangle also includes three 2-stars [25]. The statistics estimated in the model are related to the count of these structures presented in the observed network (Figure 2). To prevent the degeneracy of the model the MCMC sample size was increased up to 100,000 [26].

**2.2.3 Goodness-of-fit test and model diagnostics**

In order to check if the selected final models capture the structure of the original observed network, a set of 100 randomly generated networks were simulated using the parameters of the fitted final model. They were then compared with the observed network by four diagnostic parameters as proposed by Hunter et al. [22]:

- geodesic distance distribution defined as the proportion of pairs of nodes whose shortest connecting path is of length k, for k = 1, 2...*m* (pairs of nodes that are not connected are classified as k = 1);
- the edgewise shared partner distribution: based on the number of edges that serve as the common base for exactly *i* distinct triangles, expressing the tendency in the observed network for linked nodes to have multiple shared partners [21];

- the degree distribution or the frequency of nodes with different degree values;
- and the triad census distribution defined as the proportion of 3-node sets having 0, 1, 2, or 3 edges among them.

Frequency distributions of the four diagnostic parameters were produced for the observed data (the study network) and the 100 simulated networks. This was conducted using the built-in goodness of fit method in the package *statnet* of the statistical software R [27]. For good-fitting models, the plot of the simulated networks should closely match that of the observed network.

The statistical estimates of the parameters of the erg models indicate whether network realizations with the theoretically hypothesized properties have significantly large probabilities of being observed in subgraphs of the network data collected. Following this rationale, a further diagnostic of the final models was conducted by the following procedure: firstly, one edge of the original network was removed. Then a set of 100 randomly generated networks were simulated using the covariates of the final models fitted with new network (the original minus one edge). The number of times that the eliminated edge is included in the simulated networks was counted. This procedure was repeated by selecting randomly 10 edges present in the network and removing one at a time. Average number of times the edges are included in their respective simulated batch of one hundred networks is reported, as a measure of the reproducibility of the edge present in the observed network and subsequently removed using the covariates of the final models.

For the dyadic dependent model and in order to test its degeneracy, plots of the chain for each model statistic produced in every MCMC sample were produced. Visual exploration was

conducted to check whether the statistics of the model vary stochastically around the mean as expected in a converged model and do not depart steadily away from the mean [26]. All the analyses were conducted using the statistical software R version 2.12.0 (R Development Core Team (2010). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.R-project.org).

## 3. Results

841 responses were collected with information on either the trader or the market or both. Seventy seven responses did not include the name of the trader or farmer-trader. Of those with names, five individuals reported two markets each where they had traded and 759 only declared a single market, making a total of 764 different individuals reporting trade in the survey.

A hundred and ninety two individuals did not provide information on the number of animals traded but they provided the markets where they traded. Two did not specify market name, 194 mentioned market "none", 4 mentioned "village" as the market where they traded, making a total of 9 markets identified and 570 observations where a different pair of trader/farmer-trader and market/s could be both identified and were included in the final dataset for analysis. The 9 markets identified were: Abadale, Ankober, Arbgebeya, Chacha, Debre Berhan, Gudoberet, Keyit, Mendida and Rob gebeya. A total of 75 different villages from the 8 *kebelles* were identified in the 570 paired observations. Locations of both the villages and markets included in the analysis are shown in Figure 1.

The median number of visits to the market by traders/farmer-traders during the time window was 2 (IQR: 1-2, range 1-52). Among those who sold sheep in the markets (569), the median number

of sheep sold was 2 (IQR=1-3, range: 1-32). The most frequent reason for selling sheep was 'to buy clothes' (59%), followed by 'to buy food' (43%), 'to buy fertilizer' (35%), 'to pay taxes' (29%), 'to pay school fees' (23%) and 'to buy feed for animals' (14%). In terms of priority, these were also the reasons to sell in decreasing order of priority for the 528 respondents to this question. Among those who sold goats (50), the median number of sheep sold was 1 (IQR=1-2, range: 1-8). The traders/farmer-traders included in the survey were mainly suppliers and only 97 (17%) of them reported to have bought sheep during the reporting period (median: 2, IQR:1-2, range: 1-10). Even less individuals purchased goats (7), mostly single animals. The main reasons to buy sheep were: 'for own consumption' (50%), 'for breeding' (40%) and 'for fattening and sale' (10%).

More than a third of the 563 respondents to this question did not cross any other *kebelle* on their way to the market (39%) and when they do it, they stopped at other *kebelles* in 85% of the occasions and usually mixing with other herds 9 out of 10 times. Only 7 respondents crossed three *kebelles* (1%).

The 1-mode network contained 75 villages from 8 different *kebelles* in the Bassona Werna *wereda*. It is a dense network (42%) with a median degree of 42 (IQR: 15-53), an overall clustering coefficient of 0.37 and average geodesic distance of 1.5. These features are due to the dominant effect of the main market Debre Berhan in which traders from 54 villages (72%) operated during the reporting period. Descriptive statistics of the main village attributes are displayed in Table 1.

The negative coefficient in the initial model including only the edge count indicates fewer connections between villages in the network than would have been expected by chance ($p<0.05$).

The final dyadic independent erg model included the edge count, pairwise difference in altitude of the villages linked, distance to the main market of Debre Berhan and the second order effect of the *kebelle*, all significant at the 0.05 level. The odds of a edge where the distance to the market Debre Behran and the difference in altitude between two connected villages are both large increases significantly so that villages far away from the main market and at different altitude are more likely to be linked in the network than randomly. The odds of forming an edge between two villages in Abamote or Gudoberet adjusted by distance to Debre Berhan and altitude are approximately 75% lower than an edge between villages in any other *kebelles* ($p<0.05$ for both villages), conditional on the rest of the network, whereas edges between villages in Angolela and Bere Ager were more likely to occur than randomly and less likely between villages in Birbisa, although only significant at the alpha level of 0.1.

The final dyadic dependent erg model included the counts of edges, 2-star, 3-star and triangles configurations. Following the interpretation of the coefficients, the conditional log-odds of two villages forming a tie that is not included in a triangle, a 2-star or a 3-star is extremely low as the large coefficient of the edge count shows, increasing the odds significantly ($p<0.05$) each time the node is in one 2-star structure and decreasing it when a node is in a 3-star ($p<0.05$) or in a triangle formation ($p<0.05$). Parameters estimates and p-values as well as log likelihood and AIC of the three models are shown in Table 1.

The frequency distributions of the four diagnostic parameters of both the observed network and the 100 simulated networks for the dyadic independent and dyadic dependent models are displayed in Figure 3 and Figure 4, respectively. The independent or attribute-related model does a good job in capturing the global efficiency of the network (geodesic distances), a relative good fitting for 2 and 3 triad census, but predicts poorly the local efficiency (edge-wise shared partners) and the degree distribution due to the bimodal distribution of degree in the network whereby nodes have degree below 15 or over 50. On the other side the dyadic dependent model is able to replicate the four diagnostic parameters of the observed network much more accurately, with some predicted outliers of nodes with low edge-wise shared partners. The plots of the statistics estimated in each MCMC sample of the parameters of the dyadic dependent model are shown in Figure 5. Visually the model appears to converge with no deviation of the parameter estimators from the mean values.

The randomly removed edge appeared on average in 14% of the simulated networks for each batch in the dyadic independent model and in 15.5% in the dyadic dependent model.

## 4. Discussion

The highland town of Debre Berhan (elevation 2805 m.a.s.l.) located in the Bassona Werna *wereda* was used as a base for the research. The town is located 130 km North along the main road from the capital city of Addis Ababa. The market system observed in this community of small ruminants of Ethiopia is dominated by a large market, Debre Berhan, that serves as a meeting point for farmer/farmer-traders to buy/sell small batches of mainly sheep and at a

smaller scale goats. It lays on Road 1, one of the main arteries of the road network in Ethiopia, stretching from Addis Ababa to the border with Eritrea in the north.

Following qualitative data collected during the survey and the parameters of the observed network, most of the markets studied are medium/small scale located far from the main road, and play a secondary role in bringing in a few animals at a time which are sold onto farmer-traders or small-scale traders and then moved to other markets like Debre Berhan. These however, are often accessible by car on dirt or main roads. This centripetal general flow of live animals from production sites to larger towns is characteristic of the supply chain of livestock production in this setting [6-28-29]. The dynamism and complexity of the system reflects the opportunities to make a profit by trading with small ruminants. Traders' strategies include attending several markets each week and following a gradient of prices from the more isolated locations to larger towns and/or the capital.

It is extremely difficult to collect reliable field data on this type of settings where trade information is the main objective of the questionnaires. Not only because the unfamiliarity of the subjects to this kind of studies but also the lack of standard denomination for villages and markets alike. Despite these downsides of the data collected in this study, the analysis revealed certain patterns in the contact of production units represented by villages through the trade of small ruminants via markets.

The exponential random graph models provide a statistical framework to analyse network data by modelling the probability that any given graph is drawn from the same distribution as the observed graph. They also allow different network structures to be modelled, because the formulation is able to account for the complex structure of the network via parameters governing the entire network, rather than breaking it down into dyads [30]. Two different outputs can be extracted by fitting an erg model: the prediction of the probability of the observed overall network structure and/or the likelihood of any specific edge in an observed network. Another advantage is that the outputs of these models are interpreted in a similar manner as standard logistic regression.

The dyadic independent village-village network model shows dyadic independence because the probability of any edge does not depend on the value of or the presence of other edges, only on the attributes of the two villages (node) involved in the edge [20]. The similarity effect is strong with the distance to the main market and the difference in altitude. The edge parameter is increased/decreased to compensate the effect of the other covariates from the initial model that only contain it. This is an indicator of the density or overall cohesiveness of the network. Reading the results we conclude that there were fewer edges in this network than expected, that is, many fewer dyads of villages linked via common markets that had no other ties. In the context of the study two major constraints could be expected to influence traders/framer traders on which market to attend: distance and geographical barriers expressed in our dataset by the euclidean distance to the main market and difference in altitude from the low land to higher, respectively. Although small in the log of the odds of the edge, the difference in altitude is higher in the network than expected and so is the distance to the main market of Debre Berhan at larger scale,

which means that the geographical barriers and distance do not determine the decision on which market to trade and hence to be linked to other village of the study network. If the pattern observed was extrapolated to a larger population of small ruminant farmers, the catchment area of a market could not be estimated based on distance but on other criteria like type of market, price differential and opportunities for social interaction. In this regard Debre Berhan is on major road and the advantages of taking sheep and goats to this main market may outpower the difficulties of moving animals longer distances, from lower areas and crossing other villages contacting other flocks. The drawback of this fact is the opportunities for mixing in the way to the market.

The first model based on the attribute-related dyadic independency also showed the assortative mixing of villages by *kebelle* whereby villages within two *kebelles*, Abamote and Gudoberet, are linked less frequently than expected adjusted by distance to the main market and the difference in altitude. Although the overall effect of the network model reveals that the difference in altitude of two villages does not preclude to be linked, a potential explanation for this finding is the fact that these two *kebelles* are located in the remotest region of the *wereda* and they may tend to trade via small local markets reducing their opportunities to be linked via the larger markets identified in the study. Other attributes inherent to the 8 *kebelles* identified in the network and unaccounted for in this analysis may explain this assortative mixing.

The second model contained the dyadic dependency leading to an endogenous process of formation of ties in the form of internal structures (stars, triangles, etc.). Yet again the negative density parameter indicates that edges occur very rarely (large negative coefficient), especially if

they are not part of higher order structures such as stars and triangles. The negative triangle parameter can be interpreted as providing evidence that the edge between villages do not tend to occur in triangular structures, and hence cluster into clique-like forms. The transitive triangle parameter is an indicator of clustering and strength [31]. This statistic is interpreted as the tendency for many triangles to form together in the observed network. If high, then the model suggests regions of high triangulation indicative of core-periphery-type structures [32].

The star effects are significant suggesting that there is a tendency for multiple network partners up to degree of 2 (the positive 2-star estimate) but with a ceiling on this tendency (the negative 3-star parameter), both significant. *k*-stars are equivalent to geometrically weighted degree counts and are useful for modelling the degree distribution. In fact 1-star is equivalent to the degree of the nodes. The higher the *k*-star parameter, the easier it is for information / commodities to circulate through the network [31]. In this regard the structure of the study network showed some resilience to spread diseases globally assuming that the causative agent is mobilized via movement of small ruminants in the network.

Both models have a low reproducibility of individual edges with 14% in the attribute-related model and 15.5% in the configuration-related model. Internal structures in the network allow a better prediction of individual edges than the attributes of the node, although with a small advantage. However the dyadic dependent model predicts much better the overall structure of the network according to the four diagnostic parameters and the log likelihood of the model.

The results of the study preclude the effect of geographical barriers on the choices that traders / farmer-traders make to trade small ruminants in the study area. It could have been expected the environment to play a role in "constraining" disease transmission routes by the physical impediment of bringing animals into contact in the setting of the study. However it has been shown that the two major constraining factors, namely distance and altitude, are not deterrent for the potential contact of susceptible small ruminant populations in the Highlands of Ethiopia. It has also been observed the assortative mixing of the villages via common markets by *kebelles*. The attribute data collected at village level and included in the analysis captured a limited variability of the probability of the presence of the edge and other factors unaccounted for would definitely complement the trading criteria of the traders/farmer-traders to make their choices.

**Acknowledgements**

This study was carried out as part of the EU funded INCO-CT-2004-003670 RP/PPR MARKVAC project. We are grateful to the Ethiopian National Veterinary Institute (NVI, Debre Zeit), the farmers and farmer-traders who participated in the study and the agricultural and development officers of the Bassona Werna *wereda*. We are also grateful to Dr. Guillaume Fournie for his useful comments to the manuscript.

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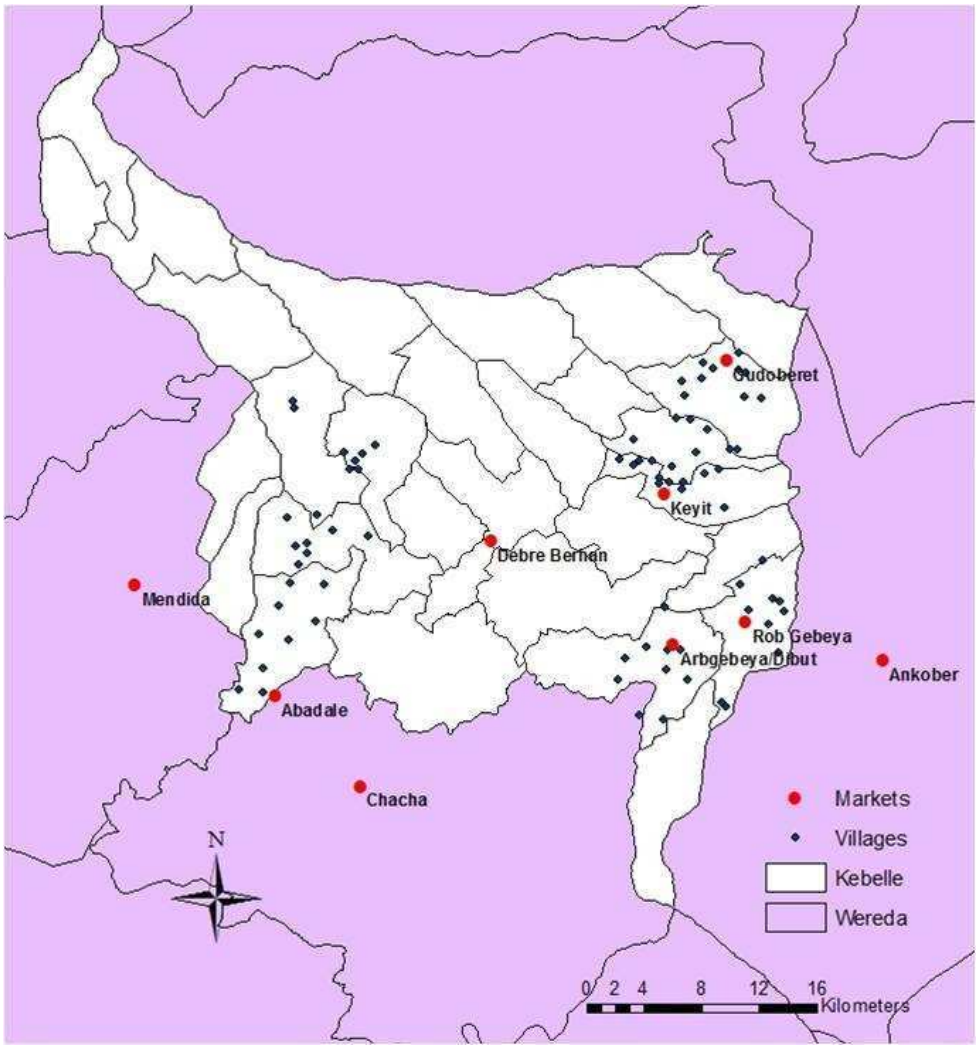
**Figure 4** Plots of the proportion of dyads against the four diagnostic parameters of both the observed networks (black) and the 100 simulated networks (grey) for the dyadic dependent model. The solid lines represent the statistics of the observed network, and the boxplots represent the distribution of 100 simulated networks based on the fitted ergm

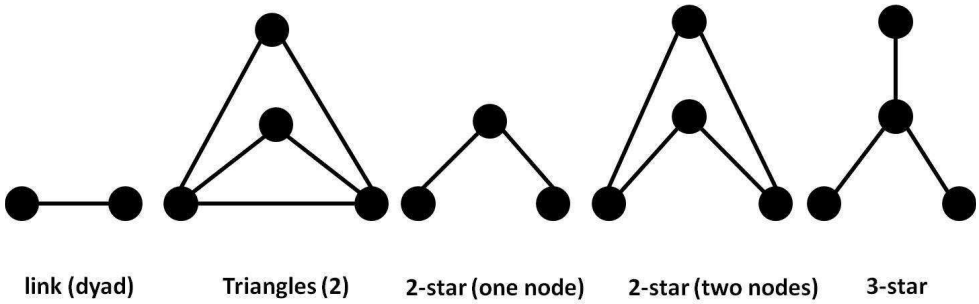
**Figure 5** Plot of the statistics estimated in each MCMC sample for the dyadic dependent (left) and the frequency histogram of the estimation of the parameters of the model (right) using a MCMC sample size of 100000.

Table 1 Descriptive statistics of the village attributes, coefficients and p values, AIC and log likelihood parameters of the three erg models: the edge count and of the final dyadic independent and dependent models

| | | Erg models | | |
|---|---|---|---|---|
| **Covariates** | **Descriptive statistics Median IQR ($25^{th}$ – $75^{th}$ )** | **Edge** | **Dyadic independent model Coefficients (P value)** | **Dyadic dependent model Coefficients (P value)** |
| Edge | | **-0.32 (<0.05)** | **-9.64 (<0.05)** | **-112.3 (<0.05)** |
| Absolute difference altitude | 3008.5 (2813-3116) | | **0.003 (<0.05)** | |
| Distance to Debre Aber | 0.13 (0.11-0.16) | | **29.9 (<0.05)** | |
| Number of small ruminant | 27 (21-34) | | | |
| Total number of sheep sold at the | 16 (12-29) | | | |
| Number of traders identified | 10 (10-10) | | | |
| Kebelle (second order effect): | | | | |
| Abamote | | | **-1.3 (<0.05)** | |
| Angolela | | | 0.6 (0.07) | |
| Bere Ager | | | 0.6 (0.07) | |
| Birbisa | | | -1 (0.08) | |
| Debele | | | -0.1 (0.73) | |
| Goshebado | | | -Inf (NA) | |
| Gudoberet | | | **-1.5 (<0.05)** | |
| Keyit | | | 0.08 (0.8) | |
| 2-star | | | | **2 (<0.05)** |
| 3-star | | | | **-0.01 (<0.05)** |
| Triangle | | | | **-1.7 (<0.05)** |
| Log-likelihood | | -1888.3 | -1418.87 | -46.9 |
| AIC | | 3778.6 | 2859.7 | 101.8 |




Location of the 75 villages and 9 markets in the Bassona Werna wereda region
116x124mm (150 x 150 DPI)




Examples of the network configurations included in the dyadic dependent exponential random graph model
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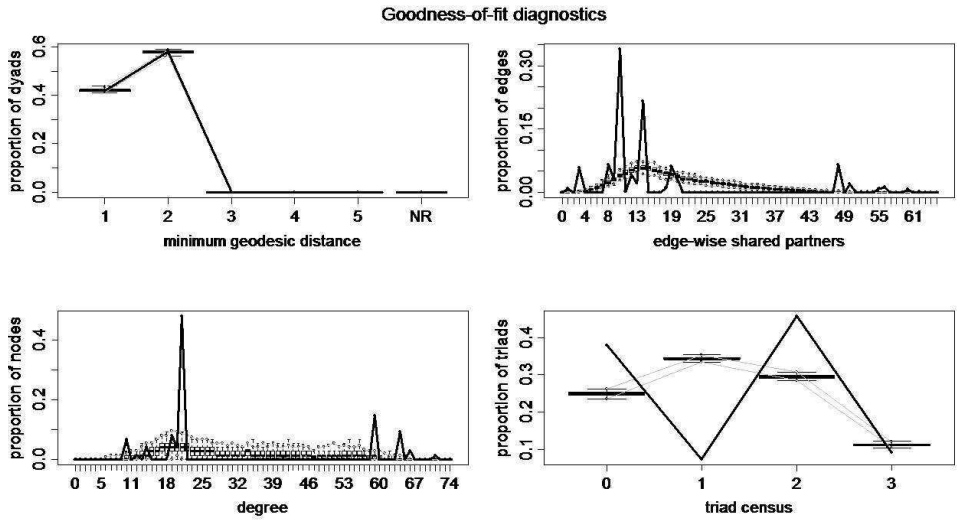
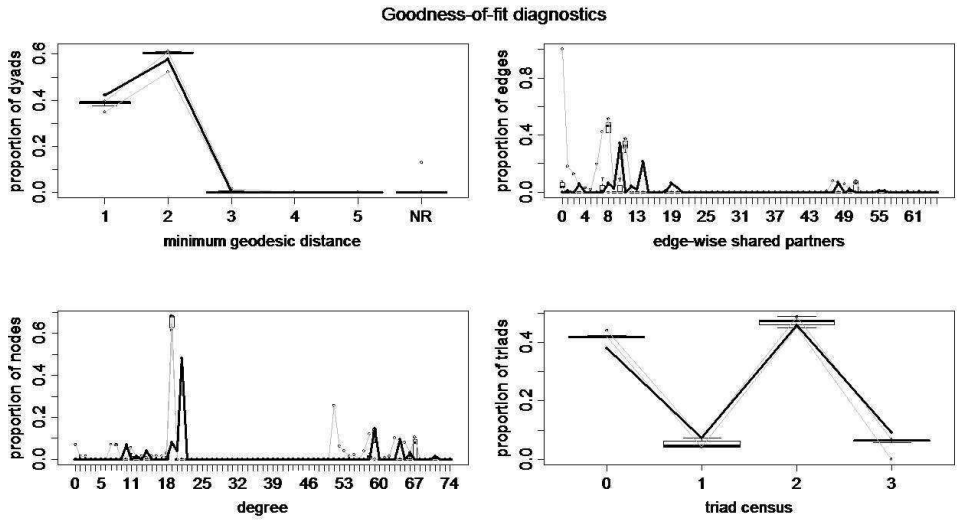



Plots of the proportion of dyads against the four diagnostic parameters of both the observed networks (black) and the 100 simulated networks (grey) for the dyadic independent model. The solid lines represent the statistics of the observed network, and the boxplots represent the distribution of 100 simulated networks based on the fitted ergm.
254x139mm (127 x 127 DPI)

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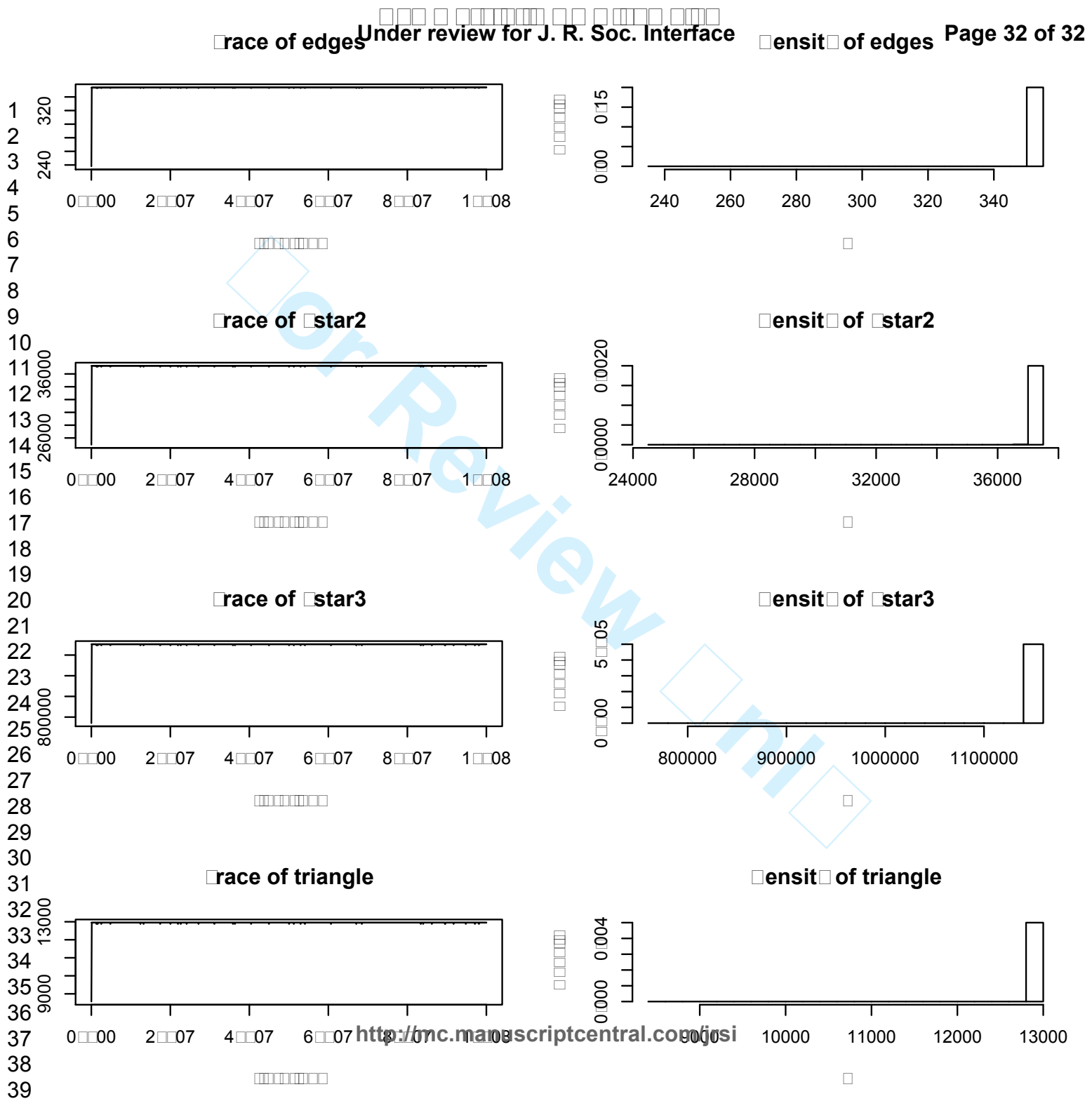
254x139mm (127 x 127 DPI)



## Annexe 2 : Cartes du nombre de foyers de PPR déclarés à l'OIE par pays et par an en Afrique entre 1996 et 2011

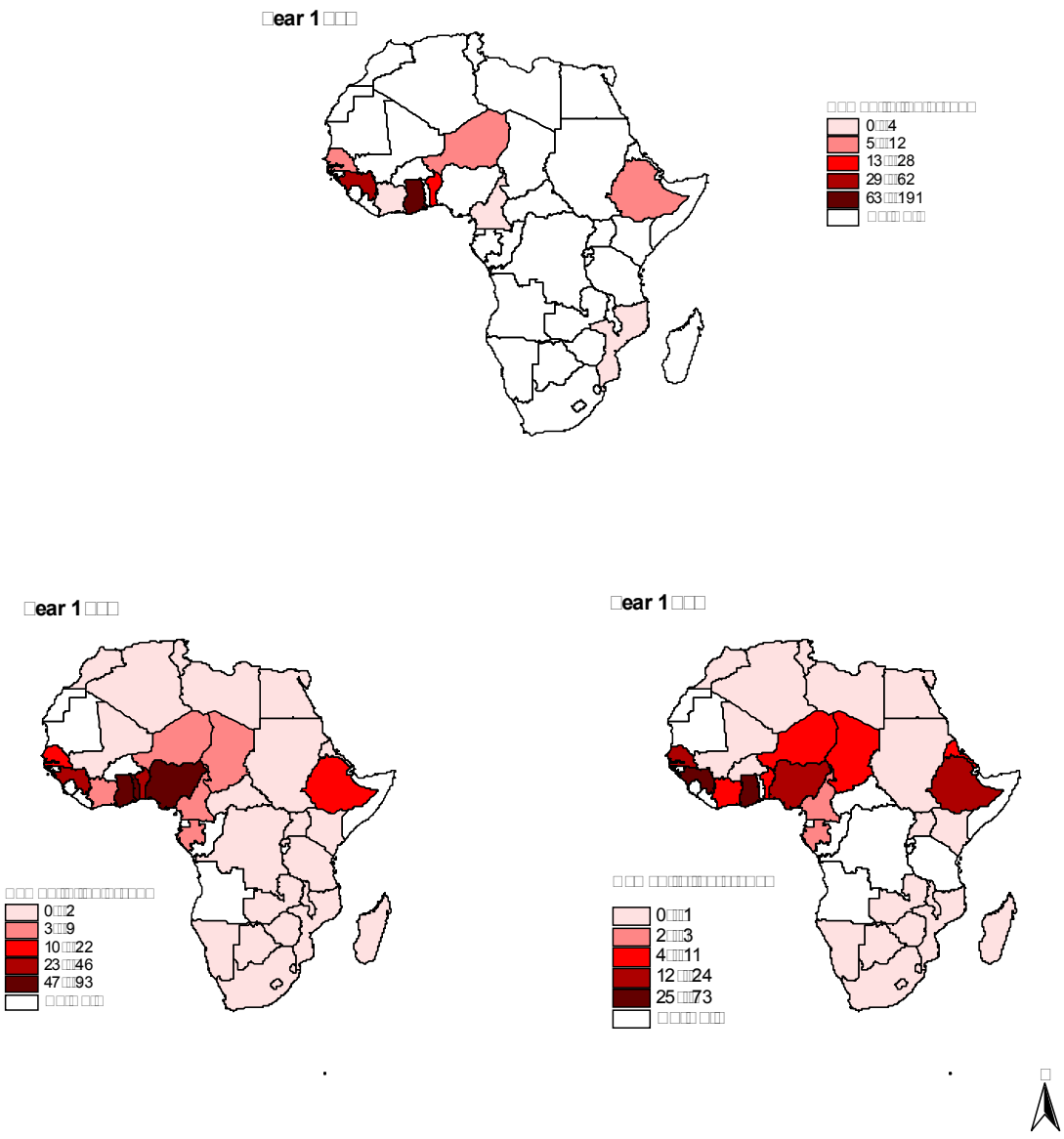



**Figure 1: Nombre de foyers de PPR déclarés par pays et par an en Afrique entre 1996 et 1998 (pendant le programme PARC, avant le PACE)**

*Source des données: OIE*

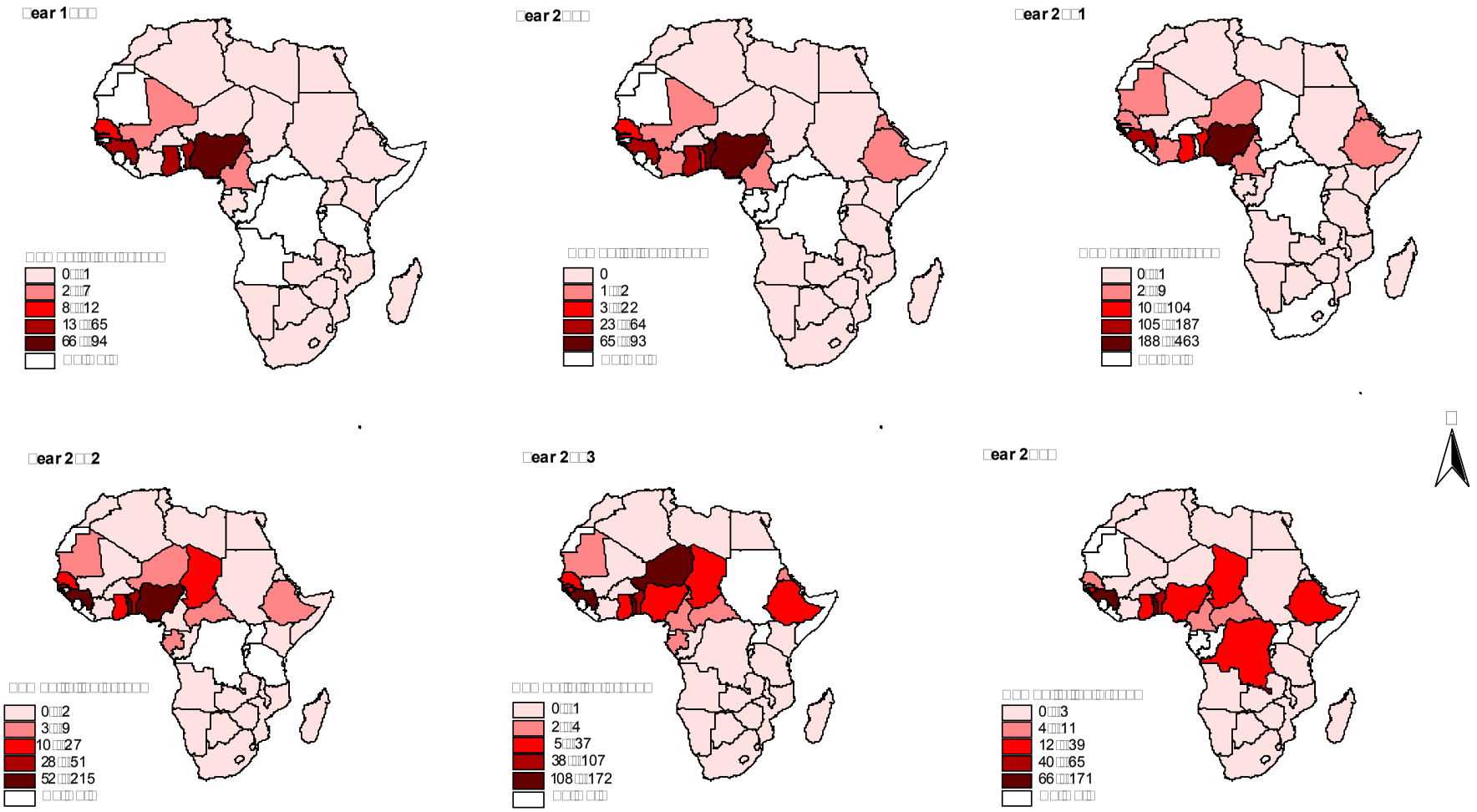



**Figure 2: Nombre de foyers PPR declares par pays et par an en Afrique entre 1999 et 2004 (pendant le programme PACE)**

*Source des données : OIE*

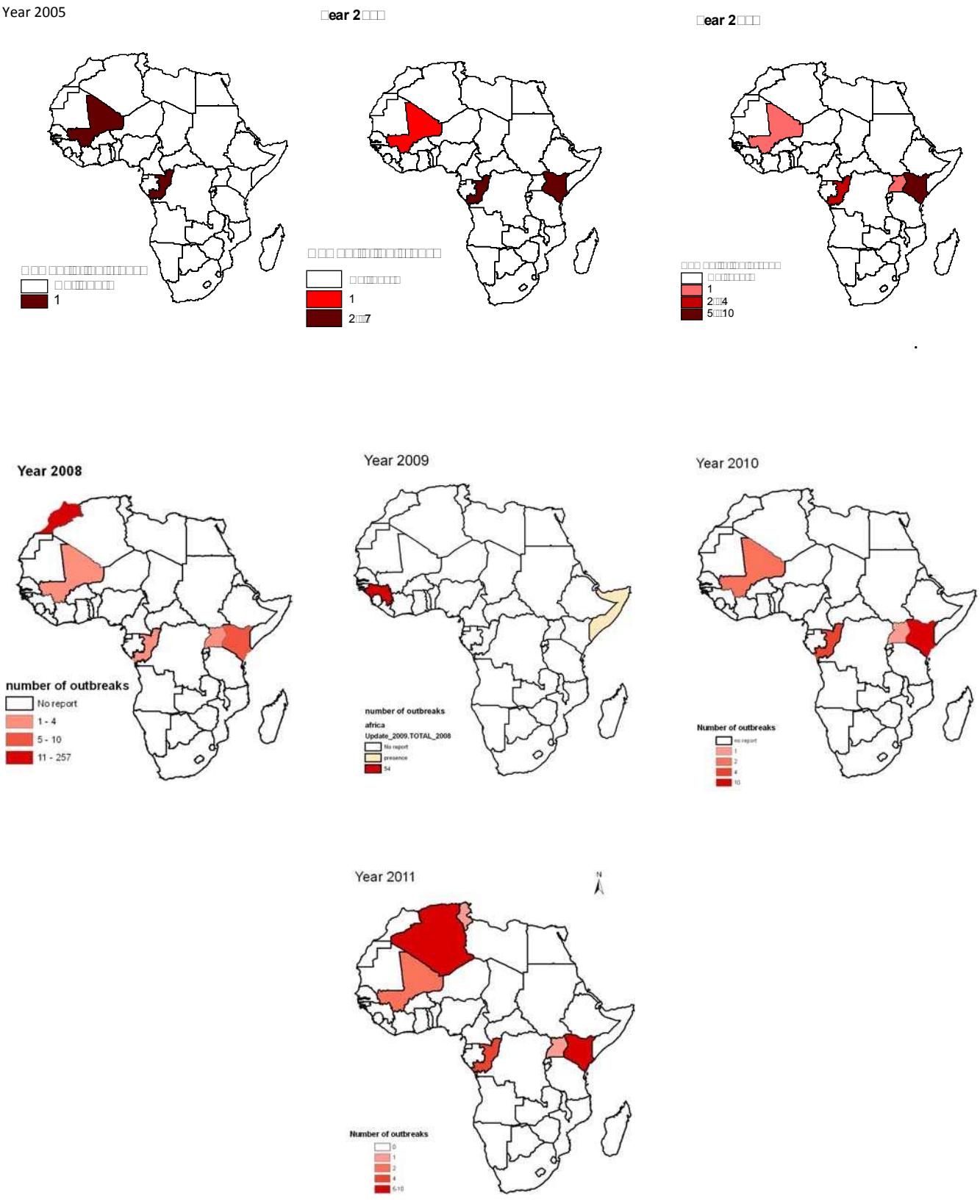



**Figure 3: Nombre de foyers de PPR declares par pays et par an en Afrique entre 2004 et 2011 (après le programme PACE).**

*Source des données : OIE*